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Quantifying the Isometric Handgrip Exercise Stimulus: Are Less Expensive Alternatives Comparable to Computerized Dynamometers?

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Quantifying the Isometric Handgrip Exercise Stimulus: Are Less Expensive Alternatives Comparable to Computerized Dynamometers?

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Abstract

Isometric handgrip (IHG) training lowers resting blood pressure (BP), but the high cost of traditional computerized IHG devices can be a barrier to use. Inexpensive mechanical IHG devices could address the cost barrier, but the acute stimulus of such devices must be determined. This study compared changes in systolic and diastolic BP, heart rate (HR), muscular activation, ratings of perceived exertion (RPE), and pain between a computerized and a mechanical IHG device during an IHG bout. Twenty healthy adults (X ± SD; Age: 22.4 ± 2.2 yrs.; ♀= 9; BP: 114/64 ± 11/7 mmHg) randomly performed an IHG bout (4, 2-min IHG contractions at 30% of maximum voluntary contraction, separated by 4-mins rest) on each device. BP, HR, and forearm surface EMG data of the non-dominant arm were collected throughout. RPE and pain were acquired at the end of each contraction.

SBP, HR, RPE and NRS-Pain scores did not differ between devices ($p > 0.05$)> However, statistically significant differences in DBP were observed ($p < 0.05$), whereby the computerized device elicited a higher DBP response than did the mechanical device. Furthermore, significant elevations in muscular activation for only the biceps brachii and extensor carpi ulnaris were observed in the mechanical handgrip device ($p < 0.05$).

The mechanical IHG induced similar acute SBP, HR, RPE and NRS-pain scores as well as similar muscular activation for 3 of the 5 muscles tested as the traditional computerized device. These findings suggest that perhaps this inexpensive alternative device could be a feasible equivalent to the traditional computerized device during acute exercise. Future studies should examine whether this inexpensive handgrip device can elicit similar training-induced reductions in resting BP as with the traditional computerized devices.

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List of Abbreviations

$[H^+]$ – Hydrogen Ion Concentration

1RM – 1 Repetition Max

ABPM – Ambulatory Blood Pressure Monitoring

ACh – Acetylcholine

ADP – Adenosine Diphosphate

AHA – American Heart Association

ABPM – Ambulatory Blood Pressure

AMP – Adenosine Monophosphate

ANP – Atrial Natriuretic Peptide

ANS – Autonomic Nervous System

AOBP – Automated Blood Pressure Device

ATP – Adenosine Triphosphate

AV – Arginine Vasopressin

BP – Blood Pressure

Ca^+ – Calcium

CC – Central Control

CCC – Cardiovascular Control Center

CCN – Cardiac Care Network of Ontario

CVD – Cardiovascular Disease

DASH – Dietary Approaches to Stop Hypertension

DBP – Diastolic Blood Pressure

E – Epinephrine

EMG – Electromyography

ET-1 – Edothelin-1

EPR – Exercise Pressor Reflex

HMP – Hypertension Management Program

HR – Heart Rate

HTN – Hypertension

IHG – Isometric Handgrip

IRE – Isometric Resistance Exercise

K^+ – Potassium

MAP – Mean Arterial Pressure

MEP – Motor End Plate

MUAP – Motor Unit Action Potential

MVC – Maximum Voluntary Contraction

Na^+ – Sodium

Na^+/K^+ Pump – Sodium Potassium Pump

NE – Norepinephrine

NO – Nitric Oxide

OBP – Office Blood Pressure

$PaCO_2$ – Partial Pressure of Carbon Dioxide

PaO_2 – Partial Pressure of Oxygen

PEH – Post Exercise Hypotension

PNS – Parasympathetic Nervous System

Pre-HTN – Prehypertension

Q – Cardiac Output

RAAS – Renin-Angiotensin-Aldosterone System

RPE – Rating of Perceived Exertion

SA – Sinoatrial Node

SBP – Systolic Blood Pressure

SNS – Sympathetic Nervous system

SV – Stroke Volume

TPR – Total Peripheral Resistance

WHO – World Health Organization

Chapter 1: Literature Review

1.1 Cardiovascular Disease

Non-communicable diseases, such as cardiovascular disease (CVD), are now the main cause of mortality worldwide (WHO 2013). CVD is an umbrella term for a group of diseases that include coronary heart disease, cerebrovascular disease, hypertension (HTN), peripheral vascular disease, heart failure, rheumatic heart disease, congenital heart disease and cardiomyopathies (WHO, 2013). CVD is the leading cause of death globally (WHO, 2013).

In 2008, CVD contributed to nearly 17.3 million deaths worldwide and is projected to reach 23.6 million deaths by the year 2030 (WHO, 2013). Nationally, nearly 62 thousand deaths/year were considered CVD-related in 2012 (Statistics Canada, 2015). More locally, CVD contributes to the deaths of nearly 1225 residents of Windsor-Essex County each year (Windsor-Essex County Health Unit, 2015).

Fortunately, advances in modern medicine have extended the lives of individuals with CVD through effective disease management and treatment. However, CVD places a substantial financial burden on the economy. In Canada, CVD was responsible for $20.9 billion dollars in direct and indirect health care expenses such as hospitalization, lost wages and disability in 2008, and healthcare-related expenses are expected to reach nearly $28.3 billion by the year 2020 (Theriault et al., 2010). Accordingly, the World Health Organization (WHO) deems CVD a global health crisis, and places an importance on primary prevention (WHO, 2013). The WHO recommends the adoption of a healthy lifestyle including the limitation of alcohol and tobacco consumption, adopting healthy dietary habits, lowering sodium intake, increasing physical activity, maintaining a healthy bodyweight and lowering high cholesterol (WHO, 2013). Recognizing the importance of preventing the progression of CVD, the government of Ontario has provided funding to programs such as the Cardiac Care Network of Ontario (CCN), one of the largest programs in North America devoted to enhancing quality, efficiency and access of

cardiovascular services in Ontario (CCN, 2017). Additionally, the Hypertension Management Program (HMP), was created to improve detection, diagnosis, treatment and prevention of HTN (Ontario Stroke Network, 2015).

1.2 Hypertension

1.2.1 Defining Hypertension

Hypertension (HTN) or chronic elevations in resting and/or ambulatory arterial blood pressure (BP), is both a type of CVD and the leading modifiable risk factor for CVD (WHO, 2013). Arterial BP is represented by two measurements: systolic BP (SBP) representing the pressure exerted onto the arterial walls when the ventricle contracts during the cardiac cycle, and diastolic BP (DBP) representing the pressure exerted onto the arterial walls during the portion of the cardiac cycle where the ventricles are relaxing, and the atria are filling (Herd, 1970).

Normal BP is indicated by a mean resting SBP of <120 mmHg and DBP of <80 mmHg (Nerenberg et al., 2018; Whelton et al., 2017; Heart and Stroke, 2017). Furthermore, measures between 120-129 mmHg SBP and or values >80 mmHg DBP are considered to be elevated or "pre-hypertensive" (Pre-HTN) (Whelton et al., 2017; Heart and Stroke; 2017; Guo et al., 2011). Pre-HTN individuals have been shown to be at greater risk of developing CVD than those with lower BP (Guo et al., 2011). Currently, HTN is classified as resting office SBP of $\geq$130 mmHg, a DBP >80 mm and/or prescribed anti-hypertensive medication (see below: Section 1.2.4; Whelton et al., 2017; Myers et al., 2015). Furthermore, individuals can be diagnosed as having HTN by using ambulatory blood pressure monitoring (ABPM) devices if they have day time BP values $\geq$135/85 mmHg, a nighttime BP >120/80 mmHg or a 24-hour average BP >130/ 80 mmHg (Nerenberg et al., 2018). HTN is further categorized into stages depending on whether a particular threshold of BP is reached. Traditionally, HTN has been defined as Stage 1 HTN as 140-159/90-99 mmHg and Stage 2 HTN as >160/100 mmHg (Nerenberg et al., 2018). Recent

Canadian and American guidelines endorse more conservative stages, with Canada identifying Stage 1 HTN as those individuals with arterial BP of 135-139/85-89 mmHg, and Stage 2 HTN as those with arterial BP of ≥140/90 mmHg (Nerenberg et al., 2018). The American guidelines offer even more conservative staging, with elevated BP ranging from 120-129/ <80 mmHg, Stage 1 HTN between 130-139/80-89 mmHg, and Stage 2 HTN ≥140/ ≥90 mmHg, Whelton et al., 2017). Furthermore, a diagnosis of hypertensive urgency HTN can be given if an individual has a BP exceeding 180/120 mmHg (Whelton et al., 2017). Finally, a diagnosis of hypertensive emergency can be given if the BP exceeds 180/120 mmHg and includes the presence of target organ damage (Whelton et al., 2017).

With advancements in BP measurement technology (see Section 1.2.4), HTN can now be more accurately diagnosed. For instance, AOBP devices provide more accurate representations of BP compared with traditional manual measurements and allow for a diagnosis of HTN to be made if an individual has a resting BP ≥135/85 mmHg (Myers et al., 2010; Nerenberg et al., 2018). New research suggests that the superior method for determining BP is through the use of ambulatory BP monitoring (ABPM), which provides insight into BP fluctuations over a 24-hour duration (Pickering et al., 2006). Individuals can be diagnosed as having HTN using ABPM devices if they have day time BP values >135/85 mmHg, a night time BP >120/80 mmHg or a 24-hour average BP >130/80 mmHg (Nerenberg et al., 2018).

HTN can also be categorized based on the origin of the elevations in BP as either primary (essential) or secondary HTN. Secondary HTN is characterized by elevations in BP that can be directly attributed to another disease such as renal failure, renovascular disease, aldosteronism and pheochromocytoma and accounts for only 5% of all HTN cases (Gupta-Malhotra et al., 2014; Carretero et al., 2000). Alternatively, primary or essential HTN has no direct cause that

can be identified but is suspected to be linked to numerous factors such as having a genetic predisposition to HTN as well as lifestyle factors such as sedentary behavior, alcohol and tobacco use, a nutritionally deficient diet, high stress and obesity (Gupta-Malhotra et al., 2014; Carretero et al., 2000). Unfortunately, primary HTN makes up 95% of all HTN cases (Gupta-Malhotra et al., 2014; Carretero et al., 2000). Consequently, prevention of HTN should be targeted through the modification of the lifestyle factors listed above (WHO, 2013).

1.2.2 Prevalence of Hypertension

Of the 17.2 million CVD-related deaths in Canada in 2012, 9.4 million of these are the result of BP-related complications due to HTN (WHO, 2013). The worldwide prevalence of HTN is steadily rising; in 2000, nearly 600 million people were diagnosed with HTN and this number rose to 1 billion by the year 2010 (Mills et al., 2016). In Canada, nearly 5.3 million Canadians were diagnosed with HTN in 2014, which equates to about 1 in every 5 Canadians as having HTN (Statistics Canada, 2016). More locally, 19% of people ages 12 years and older are diagnosed with HTN in Windsor-Essex County, which equated to nearly 74 000 people in 2011 (Windsor-Essex County Health Unit, 2014). Therefore, immediate action must be taken in primary prevention and disease management to prevent the growing rates of both HTN and CVD.

1.2.3 Blood Pressure Regulation

BP regulation is a key modulator of adequate perfusion of body tissues (Herd, 1970). BP is controlled through manipulations of cardiac output (Q) and total peripheral resistance (TPR; Raven & Chapleau, 2014). Q is the product of stroke volume (SV), and heart rate (HR; Raven & Chapleau, 2014), while TPR refers to the amount of resistance to blood flow that is exerted by the systemic blood vessels through arterial diameter manipulations known as vasoconstriction (reduced arterial diameter) and vasodilation (augmented arterial diameter; Ackerman, 2004). SV

represents the amount of blood that is pumped from the heart per beat, whereas HR refers to the number of heart beats per minute (Raven & Chapleau, 2014). Therefore, BP is altered by perturbations in Q via increases or decreases in HR and/or SV, and through manipulations of TPR via changes to arterial diameter. This process of BP modulation is controlled through the interaction of intrinsic neurological, hormonal and local arterial mechanisms.

1.2.3.1 Neurological Regulation of Blood Pressure

BP is centrally regulated by the autonomic nervous system (ANS) and its two branches: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) (Dampney et al., 2002). SNS stimulation increases the excitation of contractile neurons in the heart, which increases HR, alters Q, and results in a rise of BP (Dampney et al., 2002). The SNS can also cause vasoconstriction, subsequently increasing TPR and BP (Dampney et al., 2002). Alternatively, the PNS lowers BP by stimulating the vagus nerve which innervates the heart (Pavlov & Tracey, 2012). Vagus nerve stimulation activates the muscarinic receptors (see Section 1.2.3.2) responsible for the lowering of HR and decreasing myocardial excitability (Pavlov & Tracey, 2012; Dampney et al., 2002). Optimal BP is maintained through balancing the stimulation of the SNS and PNS.

The activity of the PNS and SNS is regulated by two higher control centres in the brain: the central command (CC) and the cardiovascular control centre (CCC) (Nobrega et al., 2014). The CC senses and regulates ANS activity via stimulation of the CCC (feed-forward control) that innervates the heart and blood vessels, which can result in either SNS or PNS stimulation to increase or decrease BP, respectively (Nobrega et al., 2014). Additionally, this system also has innate negative feedback control through receptors located in the periphery known as arterial baroreceptors, chemoreceptors, and muscle afferent receptors. These receptors all provide feedback to the CCC to maintain a particular BP "set point" (Nobrega et al., 2014).

Arterial baroreceptors are sensitive to tensile changes in the carotid arteries, pulmonary vessels, and aorta (Lafranchi & Somers, 2002). When BP is increased above a certain set point, this causes pressure to be placed on the walls of the blood vessel, resulting in stretching of the arterial walls (Papaioannou, 2007). Baroreceptors detect this stretching and trigger afferent signalling to the CCC, which in turn decreases SNS stimulation, HR, cardiac contractility and TPR (Papaioannou, 2007). Alternatively, if BP drops below the set point, a decrease in tension against the vessel walls triggers afferent signalling from the baroreceptors to the CCC (Papaioannou, 2007). These signals reduce PNS and increase SNS signalling to increase HR, cardiac excitability, and TPR in order to raise BP back to the homeostatic set point (Papaioannou, 2007; Lafranchi & Somers, 2002).

Chemoreceptors, located in the carotid and aortic arteries, are responsible for detecting perturbations of the partial pressures of oxygen (PaO_2), carbon dioxide ($PaCO_2$), and hydrogen ion concentrations ($[H^+]$) that are produced as a result of increased cellular respiration (Moreira et al., 2011). Under low oxygen states, such as during moderate to vigorous aerobic exercise, chemoreceptors detect a decrease in $[PaO_2]$ in addition to an increase in $[PaCO_2]$ and $[H^+]$, which triggers afferent signalling to the CCC to stimulate SNS activity (Moreira et al., 2011). This stimulation of the SNS raises HR, Q and TPR, which increases BP and stimulates ventilation for the purposes of adequate tissue perfusion of oxygen (Moreira et al., 2011).

The final periphery receptors involved in the feedback control between the CC and CCC are the muscle afferent receptors. There are two types of muscle afferent receptors that regulate BP homeostasis: type III afferent mechanoreceptors and type IV metaboreceptors (Leshnower et al., 2001). Similar to arterial baroreceptors, the main function of the type III afferent mechanoreceptors is to sense stretch and pressure differences within the arteries that occur

during muscular contraction (Leshnower et al., 2001). When stimulated, type III afferent mechanoreceptors relay this information back to the CCC, influencing SNS stimulation and PNS inhibition, which raises Q through increases in HR and subsequently raises BP (McCord & Kaufman, 2010). Although mechanoreceptors and metaboreceptors serve a similar purpose (alter BP in response to physical demands such as exercise), type IV afferent metaboreceptors differ in terms of the stimuli to which they are sensitive. Metaboreceptors are receptors that are sensitive to increases in the concentration of by-products of metabolism, which include lactic acid, potassium (K^+), deprotonated phosphate, serotonin, bradykinin and adenosine (McCord & Kaufman, 2010). When elevated concentrations of metabolites are detected, the metaboreceptors relay this information to the CCC, which augments SNS activity, reduces PNS stimulation and increases Q, HR, and TPR, ultimately increasing BP to clear these metabolic by-products from metabolically active tissue (McCord & Kaufman, 2010). This increase in BP allows for greater perfusion of blood to the active muscle, thus delivering more O_2 to active tissues and facilitating the disposal of accumulated metabolites (McCord & Kaufman, 2010). Mechanoreceptors and metaboreceptors ultimately work synergistically to accommodate for normal hemodynamics during exercise (McCord & Kaufman, 2010).

1.2.3.2 Hormonal Regulation of Blood Pressure

Changes in BP can also be regulated via the neuroendocrine system through coordinated actions of hormones (Gordon et al., 2015). The hormones influencing BP include the catecholamines epinephrine (E) and norepinephrine (NE), acetylcholine (ACh), angiotensin, aldosterone, arginine vasopressin (AVP) and atrial natriuretic peptide (ANP).

The ANS is regulated by preganglionic and postganglionic neurons that secrete neurotransmitters at the synaptic clefts to transfer signals across the axons (Thomas, 2011). The neurotransmitter ACh is released between the preganglionic and postganglionic neurons in both

the SNS and PNS, however the SNS releases NE via postganglionic neurons at the synaptic cleft, whereas the PNS releases ACh (Gordan et al., 2015). Both E and NE are secreted by the adrenal medullary cells via stimulation of sympathetic innervation (Thomas, 2011). The function of E and NE is mainly dependent on the type and location of the receptor to which they bind (Guimaraes & Moura, 2011). There are two main types of receptors: α-adrenergic and β-adrenergic (Guimaraes & Moura, 2001). The α_1-adrenergic receptors are found in most vascular smooth muscle of sympathetic target organs (excluding the heart), while the α_2-adrenergic receptors are located near the synaptic junction of SNS nerve cells of vascular beds (Guimaraes & Moura, 2001). Activation of both types of α-adrenergic receptors elicit vasoconstriction (Thomas, 2011). β_1-adrenergic receptors are found in the heart, kidneys, lungs, and adipose tissue, whereas β_2-adrenergic receptors are found in most SNS target organs such as the heart, eyes, kidney, brain, gastrointestinal tract, and vascular smooth muscle (Guimaraes & Moura, 2001; Gordon et al., 2015). Stimulation of β_1-adrenergic receptors by E or NE leads to increases in HR and myocardial contractility, as well as triggering the kidneys to release renin to increase vasoconstriction and ultimately BP (Gordon et al., 2015). β_2-adrenergic receptor activation causes vasodilation of SNS target organs such as the heart, liver and skeletal muscles in order to increase blood perfusion to metabolically active tissue (Guimaraes & Moura, 2001). The activation of α-adrenergic and β-adrenergic receptors causes changes in smooth muscle diameter via vasoconstriction and vasodilation, ultimately resulting in the regulation of BP from changes in TPR (Furchgott, 1983).

Alternatively, the PNS is regulated by the release of ACh at the synaptic junction of parasympathetic nerve cells (Gordon et al., 2015). The binding of ACh occurs on two types of muscarinic receptors known as M2 and M3 receptors (Gordon et al., 2015). M2 muscarinic

receptors are found in abundance in cardiac muscle cells. ACh binding triggers M2 receptor activation, resulting in decreasing depolarization through perturbations in conduction velocity across the atrioventricular node (Gordon et al., 2015). This works to synergistically promote normal hemodynamics through a decreased cardiac contractility through a reduction in HR, subsequently leading to decreases in Q and BP (Gordon et al., 2015; Mysliveček & Trojan, 2003). M3 receptor activation facilitates the release of nitric oxide (NO) from the endothelium which causes vascular vasodilation and a decrease in BP (see Section 1.2.3.3; Brodde & Michel, 1999).

The renin-angiotensin-aldosterone system (RAAS) works to regulate BP through its influence on vascular tone and blood volume (Nguyen et al., 2002). Reductions in blood volume triggers the release of the glycoprotein renin from the kidneys, which converts the non-active pro-hormone angiotensinogen into angiotensin I (Nguyen et al., 2002). Angiotensin I is further modified by angiotensin converting enzyme (ACE) into angiotensin II, the active form of the hormone (Nguyen et al., 2002). Angiotensin II is a powerful vasoconstrictor that increases BP through increases in TPR (Nguyen et al., 2002). Decreases in blood fluid volume also trigger the release of a hormone known as aldosterone from the adrenal cortex, which stimulates fluid retention through conservation of sodium (Granger & Schnackenberg, 2000). This process increases the blood fluid volume, which raises SV and increases BP via increases in Q (Weir & Dzau, 1999; Hall et al., 1990).

AVP, also known as antidiuretic hormone, is secreted by the posterior pituitary in response to decreases in blood fluid balance and acts as a powerful vasoconstrictor that increases BP by augmenting TPR (Henderson & Bryon, 2007; Nguyen et al., 2002). AVP works particularly well at stimulating vasoconstriction in skin, muscle, and visceral arterial beds

(Henderson & Bryon, 2007). AVP also stimulates the release of aldosterone, which causes an increase in blood volume, SV, and BP (Granger & Schnackenberg, 2000; Hall et al., 1990).

ANP is a hormone secreted by the atria in response to increased stretching of the atrial vessel walls in the heart (Venugopal, 2001). Release of ANP is related to increased sympathetic activation (Ruskoaho, 2011). Therefore, ANP lowers BP by inhibiting actin-myosin binding of cardiac vessel smooth muscle, which inhibits vasoconstriction of the blood vessels and lowers TPR (Münzel et al., 2003). ANP can also control BP through decreases in blood volume. Release of ANP triggers the kidneys to expel both water and sodium, leading to decreases in Q through a smaller SV, ultimately reducing BP (Venugopal, 2001).

1.2.3.3 Local Blood Pressure Control

BP is regulated through locally secreted substances in the peripheral vasculature that are produced in response to metabolic demands of tissues. These locally released substances include NO, endothelin-1 (ET-1), K^+, and adenosine derivatives; they work by modifying BP through changes in TPR via alteration of the diameter of smooth muscle vasculature (Webb, 2003).

The increase in blood flow to muscles is associated with an increased metabolism, which elicits a rise in the amount of vascular pressure or “shear stress” exerted against the most inner layer of the blood vessel known as the endothelium (Furchgott, 1983). This increase in endothelial pressure triggers the release of a potent vasodilatory compound known as NO (Furchgott, 1983). NO is derived from the amino acid L-arginine by the enzyme NO synthase (Thijseen et al., 2011). NO then diffuses out of the endothelium where it can react with vasculature smooth muscle to increase arterial diameter, reducing TPR and thus lowering BP (Paniagua et al. 2001).

ET-1, a vasoconstrictor, is a regulatory mechanism for BP that counters the localized NO, as previously noted. ET-1 production is activated by endothelial chemicals (AVP and NE) and mechanical (shear stress) stimulation (Agapitov & Haynes, 2002). The action of ET-1 is entirely dependent on which type of ET-1 receptor is activated. Upon activation, ET_{B2} and ET_A receptors trigger vasoconstriction of vascular smooth muscle, thus increasing TPR and raising BP (Gordon et al., 2015). Alternatively, ET_B receptor activation causes NO release from the endothelium due to increases in shear stress, triggering vascular smooth muscle relaxation (vasodilation), resulting in decreased TPR and ultimately lowering BP (Agapitov & Haynes, 2002).

K^+ is another key player in localized BP regulation (Haddy et al., 2006). During muscular contractions, K^+ is released from the endothelium due to sheer stress and accumulates in the intracellular space of vascular smooth muscle membranes (Haddy et al., 2006). This accumulation of K^+ stimulates the Na^+/K^+ pump to hyperpolarize the cell, which in turn inhibits the influx of Ca^+ (Haddy et al., 2006). This decrease in intracellular Ca^+ inhibits the ability of vascular smooth muscle to contract, which lowers TPR, and thus decreases BP (Haddy et al., 2006).

Adenosine triphosphate (ATP), an adenosine derivative, is an important substrate for muscular contraction (Marshall, 2007). Utilization of ATP in muscular contraction elicits the accumulation of ATP by-products, such as adenosine diphosphate (ADP), adenosine monophosphate (AMP), and adenosine (Haddy & Scott, 1968). As ATP concentrations decrease and the concentration of other adenosine metabolites increase, these metabolites activate adenosine receptors on the endothelium known as A_1, A_{2A} or A_{2B} receptors (Marshall, 2007). Activation of these receptors generates a vasodilatory effect on smooth muscle, which increases

the arterial diameter to augment blood flow to the working muscles in order to combat the metabolic demand (Marshall, 2007). This vasodilation decreases TPR, and thus lowers BP.

1.2.4 Blood Pressure Measurement

BP measuring techniques range in level of invasiveness and accuracy. Currently, the most accurate method of BP measurement involves the implantation of a catheter into the radial artery (Balaji & Shah, 2011). Beat-to-beat SBP, DBP and mean arterial pressure (MAP; [systolic BP + 2(diastolic BP)] / 3) are derived from a pressure transducer attached to the catheter, which is inserted into the brachial artery through the wrist (Balaji & Shah, 2011). Although this method is classified as the gold standard in BP measurement, due to the higher costs, invasiveness and expertise required to administer the catheter, it is not used as a practical BP measurement modality by most clinicians (Balaji & Shah, 2011). Currently, clinicians can implement less invasive, time- and cost-effective measurement tools for BP like auscultatory sphygmomanometry and oscillometry (Pickering et al., 2005). Both techniques are non-invasive and obtain measures at the level of the brachial artery (Pickering et al., 2005). Non-invasive forms of BP monitoring provide similar accuracy to invasive techniques (Bing et al., 2014). Furthermore, there is growing clinical interest regarding a novel form of oscillometry known as "ambulatory BP monitoring". Ambulatory BP monitoring allows for BP measurement over a 24-hour duration and evidence suggests it is a better prognostic indicator of CVD compared to office BP measurement (Sherwood et al., 2012).

1.2.4.1 Auscultatory Sphygmomanometry

A non-invasive technique known as auscultatory sphygmomanometry is performed by utilizing an inflatable cuff and sphygmomanometer to determine BP (Beevers et al., 2001). BP is determined through the timing of audible sounds produced as blood flow causes oscillations inside arterial walls known as "Korotkoff sounds" (Beevers et al., 2001). This technique is

employed when assessing "office BP" (OBP; Nerenberg et al., 2018). An inflatable cuff is placed around the patient's arm and inflated to a point at which no Korotkoff sounds are heard due to occlusion of blood flow (>SBP; Pickering et al., 2005). A stethoscope is then placed at the point of radial pulsation (Beevers et al., 2001). The cuff is gradually deflated and the examiner listens for the onset and absence of the Korotkoff sounds (Beevers et al., 2001). The Korotkoff sounds can be categorized into 5 phases: the 1st phase is categorized by the first presence of Korotkoff sounds and represents SBP, while the 5th stage is when the audible sound is no longer heard and represents DBP (Pickering et al., 2005).

Although this technique is non-invasive, and at one point was widely used among physicians, it has inherent limitations due to its dependence on the skill of the observer to interpret the exact onset and offset of the Korotkoff sounds (Pickering et al., 2005). Lack of concentration, poor hearing and confusion from visual or audible cues have been reported to cause error in BP measurement using this technique (Beevers et al., 2001). Furthermore, observer bias has been suggested to also be a limitation to this method, as the observer may already have a preconceived notion of what an individual's BP may be based on their physical appearance, sex or age (Beevers et al., 2001). Observer bias may lead to an under-diagnosis of BP for younger healthy weight individuals and over-reporting BP for older or obese individuals (Beevers et al., 2001). Another common limitation to using this technique occurs due to assignment of the wrong size cuffs for individuals (Manning et al., 1983). Using a cuff that is too big leads to an under-estimation of reported SBP and DBP values by 10-30 mmHg due to these cuffs requiring more pressure to occlude arterial blood flow (Manning et al., 1983). Due to these limitations, the auscultatory sphygmomanometry method could fail to diagnose or misdiagnose HTN (Pickering et al., 2005).

1.2.4.2 Oscillometry

Another non-invasive BP measurement technique using an inflatable cuff employs "oscillometry" to derive BP, and is used for AOBP measurement (Nerenberg et al., 2018). Unlike auscultatory sphygmomanometry, oscillometry does not rely on human interpretation of Korotkoff sounds, but instead measures BP via an electric pressure transducer (Stergiou et al., 2011). As with auscultatory sphygmomanometry, a cuff is inflated over the arm until no oscillations are detected by a pressure transducer within the device (Ogedegbe & Pickering, 2010; Berger, 2001). As the cuff is gradually deflated, the pressure transducer detects the point of maximal oscillation, which has been shown to correspond with MAP (Ogedegbe & Pickering, 2010). The oscillometric device can then calculate SBP and DBP using algorithms (Pickering, 2005). The main advantage to this technique over the auscultatory sphygmomanometer is that it prevents observer bias and error, as well as being less influenced by external noise (Stergiou et al., 2011). However, a notable limitation to this technique is that the oscillometric algorithms used to determine BP do not consider external factors influencing arterial BP like arterial stiffness, which can lead to errors in calculation of BP (Lui et al., 2013). It is also important to note that body position can influence BP measurement; participants should be seated with legs uncrossed and with the arm supported in a position that is approximately heart height (Pickering et al., 2005). Furthermore, it is important to ensure that the participant is fitted with an appropriately sized cuff, as cuffs that are too large or small have been shown to lead to inaccurate measurements of BP (Pickering et al., 2005). In addition, the algorithms used to derive BP are not standardized and vary depending on manufacturers, which could lead to potential discrepancies in BP values obtained between devices (Ogedegbe & Pickering, 2010). Despite these potential limitations, evidence supports oscillometry as providing reliable representation of BP, when compared to auscultatory sphygmomanometry (Stergiou et al., 2011).

1.2.4.3 Ambulatory Blood Pressure

ABPM is another oscillometric technique that involves acquiring BP measurements across a 24-hour time span (O'Brien et al., 2013). Unlike other oscillometric devices, the inflatable hose is attached to a portable monitor, which allows individuals to have BP measurements taken in non-clinical settings that may more accurately represent BP (O'Brien et al., 2013). The ABPM device is programed to take BP measurements every 30 minutes, with some protocols even utilizing BP measurement every 15 or 20 minutes (O'Brien et al., 2013; Pickering et al., 2006). The data from the ABPM device are transferred to a computer where a mean daytime, nighttime and 24-hour BP values can be determined (O'Brien et al., 2013; Pickering et al., 2006).

The main advantage of using ABPM is that its portability allows BP to be measured in non-clinical settings and prevents the phenomenon known as "white coat hypertension" (O'Brien et al., 2013, Franklin et al., 2013). White coat HTN is defined as BP measurements within the HTN range when BP is examined in a clinical setting, but values fall within the normal range when BP is taken at home or a non-clinical setting (Franklin et al., 2013). White coat HTN is suggested to be caused by anxiety in a clinical setting and is particularly prevalent in women, older adults and those recently diagnosed as having HTN (Franklin et al., 2013). ABPM can be used to rule out white coat HTN, and therefore, prevent misdiagnosis of HTN, and potentially limit unnecessary treatment of non-hypertensive individuals (i.e., being prescribed anti-hypertensive medications) (Lovibond et al., 2011).

In healthy individuals, SBP and DBP decreases or "dips" at least 10% during sleep; these individuals have been classified as "normal dippers" (Birkenhäger & van den Meiracker 2007; Mahabala et al., 2013). ABPM can be used to assess cardiovascular risk by measuring BP throughout the sleep cycle to determine whether an individual expresses normal dipping of BP

(>10% reduction in BP), or has reductions <10%, which would classify them as a non-dipper (Birkenhäger & van den Meiracker 2007; Mahabala et al., 2013). Furthermore, dipping >20% would classify an individual as an excessive dipper (Kazuomi et al., 1996). Evidence supports that non-dipping and excessive dipping are correlated to increased cardiovascular risk, such as increased arterial stiffness, cardiac organ damage, diabetic retinopathy, impaired glucose tolerance and increased risk of CVD development (Hermda et al., 2013; Birkenhäger & van den Meiracker 2007; Dela Mea et al., 2005; Kazuomi et al., 1996). Additionally, ABPM has been shown to be a better predictor of CVD-related mortality and morbidity in women than men (Boggia et al., 2011). Therefore, ABPM is a useful tool for both the diagnosis of HTN, and the determination of CVD risk for individuals diagnosed with HTN.

1.2.5 Pathophysiology of Hypertension

Although the direct mechanism of primary HTN development is not entirely understood, evidence supports the idea of pathologically high BP resulting from perturbations to neural, hormonal and/or local homeostatic mechanisms (Bakris & Mensah, 2002).

The ANS plays a key role in BP maintenance. In the case of HTN, evidence suggests that overstimulation of the SNS leads to increases in BP through unusually high concentrations of circulating adrenergic neurotransmitters (NE and E) in hypertensive individuals (Mancia & Grassi, 2014; Ferrier et al., 1993). This increase in stimulation of sympathetic nerves and leads to increases in Q due to elevated HR and fluid retention that will ultimately increase BP (Parati & Esler, 2012). Along with SNS overstimulation, under-activation of the PNS is observed in hypertensive individuals, which leads to reduced vagus nerve inhibition of HR (Mancia & Grassi, 2014). This combined overstimulation of the SNS and under-activation of the PNS leads to increases in Q and ultimately BP (Olshansky et al., 2008).

In the case of HTN, chronic elevations in BP lead to structural remodelling of the arterial walls, which may ultimately cause the stiffening of cardiac and peripheral blood vessels (Mayet & Hughes, 2003). Recall that baroreceptors located in the carotid, pulmonary and aortic blood vessels detect mechanical stretching of the vessel, and once a threshold of stretch is detected, they will trigger CC signalling to shunt SNS activity and ultimately lower BP (Lafranchi & Somers, 2002). In the case of HTN, chronic elevations in BP cause vascular remodelling of the carotid arterial walls causing them to thicken and loose elasticity (Honzikova & Fiser, 2009). Thus, baroreceptors lose sensitivity and become less able to detect stretching of the carotid arteries, which can lead to chronically increased BP (Honzikova & Fiser, 2009). This effect of baroreceptor insensitivity is supported through research showing that individuals with chronically high BP have decreased baroreceptor function, compared to individuals with normal BP (Mussalo et al., 2002).

In regard to the RAAS system, the increased SNS activation discussed above leads to elevations in circulating renin and angiotensin II, leading to increased fluid retention and vasoconstriction, ultimately increasing BP (Manrique et al., 2009). There is growing evidence to support that this increase in RAAS activity negatively affects vasculature structure remodelling, which contributes to systemic stiffening of the arteries and cardiac and peripheral blood vessels, which increases TPR, BP and lowers baroreceptor sensitivity (Pacurari et al., 2014). Additionally, in cases of endothelial dysfunction, production of vasoconstrictor ET-1 is up-regulated and NO is down-regulated (Hayes & Webb, 1998). This leads to localized vasoconstriction and impaired vasodilation, which increases TPR, and in turn, BP (Hayes & Webb, 1998). There is evidence to show that hypertensive individuals display higher concentrations of ET-1, compared to a normotensive control group (Shichiri et al., 1990). This

elevation in ET-1, together with limited NO production, leads to impaired local control of vasculature, which increases TPR and ultimately BP (Dharmashankar & Widlansky, 2010).

There are numerous lifestyle factors that are thought to contribute to chronic elevations in BP. Excessively high sodium intakes, lack of physical activity, high psychological stress, smoking, diet lacking in fruits, vegetables, magnesium, Ca^+ and K^+ have been shown to increase the development of HTN (Nerenberg et al., 2018; Frisoli et al., 2011).

The risk of developing HTN also increases as a result of aging (Sun, 2015). For example, arterial stiffening throughout the aging process is suggested to be caused by a combination of metabolic syndrome, inflammation and dysfunction of neural hormonal pathways (i.e., increased SNS activity, aldosterone production and Na^+ sensitivity), which all can contribute to endothelium dysfunction and increased arterial stiffness (Sun, 2015). Additionally, aging is associated with decreased baroreceptor function (Webber et al., 1989). Increased sodium retention, arterial stiffening and decreased baroreceptor function can all lead to increases in TPR (Sun 2015; Webber et al., 1989). This increase in TPR and Q leads to elevations in BP in elderly individuals (Sun, 2015; Webber et al., 1989).

Sex differences occur in BP regulation. A meta-analysis examining resting BP across the sexes found that up to menopause, women have significantly lower BP compared to men aged 18-39 years (Sandberg & Ji, 2012). Furthermore, white non-Hispanic women aged 50-69 years still had lower BP than men, but sex differences became less apparent for this age cohort for non-Hispanic black and Mexican American individuals (Sandberg & Ji, 2012). Concernedly, women over the age of 70 years had higher instances of HTN than men of the same age (Sandberg & Ji, 2012). Although the exact mechanisms of why pre-menopausal women have lower BP compared to men requires further investigation, evidence suggests that estrogen elicits protective

cardiovascular effects in women that contribute to better endothelial function, and consequently, reduced TPR and BP (Murphy & Kelley, 2011).

Finally, there are racial and ethnic differences in BP. In 2006, Wang and colleagues examined the differences in ambulatory BP of European Americans and African Americans from childhood to early adulthood and found that African American individuals had higher day-time and night-time ambulatory mean SBP and DBP compared to European Americans (Wang et al., 2006). Furthermore, African Americans showed significantly less night-time BP dipping than the European Americans, which was further exasperated as the participants aged (Wang et al., 2006). As discussed in the ABPM section above, non-dipping is associated with increased risk of CVD (see Section 1.2.3 Blood Pressure Measurement). Findings from Wang and colleagues (2006) would suggest that African American individuals are at increased risk of developing CVD and other HTN-related complications, such as organ damage, compared to their European American counterparts (Wang et al., 2006; Sega et al., 2002).

1.2.6 Hypertension Treatment and Management

Ultimately, the aim of HTN management is to lower elevated BP into ideal clinical ranges (Pescatello et al., 2015). Currently, the Canadian Hypertension Education Plan recommendation is for hypertensive individuals to reduce SBP <135 mmHg and DBP <85mmHg obtained through AOBP devices (Nerenberg et al., 2018). Interestingly, the American guidelines have made new recommendations to reduce BP below SBP <120 mmHg and DBP <80 mmHg, showing that the recommendation in this field are continually changing (Whelton et al., 2017). Additionally, individuals who are at high risk of developing CVD (without diabetes or stroke) require more intensive reduction in SBP of <120 mmHg to reduce risk of CVD development (Nerenberg et al., 2018). Additionally, it is recommended that Pre-HTN individuals reduce their

resting AOBP SBP to <120 mmHg and/or DBP <80 mmHg to prevent the advancement of HTN (Rosendorff et al., 2007). Elderly patients (≥80 years of age) are recommended to lower SBP below 150 mmHg (Nerenberg et al., 2018). It is recommended to lower 24-hour ambulatory SBP and DBP to <130/80 mmHg, daytime ambulatory SBP and DBP to <135/85 mmHg, and night time SBP and DBP to ≤ 120/70 mmHg, respectively (O'Brien et al., 2013).

Recently, evidence from the SPRINT research group examining the effects of reducing BP of adults 50 years and older with SBP ranging from 130-180 mmHg to either <120 mmHg (intensive reduction group) or <140 mmHg (traditional reduction group), found that reducing SBP to below 120 mmHg resulted in fewer instances of fatal and nonfatal major cardiovascular events from any causes, compared to the <140 mmHg group (SPRINT research group; 2015). A recent meta-analysis (123 studies comprised of 613 815 patients undergoing BP reducing trials) conducted by Ettehad and colleagues (2015) examined the effects of SBP reductions and its relation to the cases major cardiovascular events (Ettehad et al., 2015). Researchers found that every 10 mmHg reduction in SBP resulted in a proportional decrease in major CVD events, coronary heart disease, stroke, heart failure, and all-cause mortality (Ettehad et al., 2015). These studies provide support for the importance of lowering SBP in hypertensive individuals.

Currently, management of HTN is achieved through either lifestyle modification, and in many cases, the addition of pharmacotherapies (Nerenberg et al., 2018; Whelton et al., 2017). Adhering to proper dietary habits and engaging in regular exercise are key in the prevention and treatment of HTN (Nerenberg et al., 2018). Other recommendations for the management of BP are to limit alcohol consumption to ≤2 drinks per day, to maintain a healthy bodyweight, reduce stress, reduce sodium consumption and to cease smoking (Nerenberg et al., 2018). Dietary modifications targeting the management of HTN are summarized in the Dietary Approaches to

Stop Hypertension (DASH) diet, which emphasizes the consumption of fruits, vegetables, low-fat dairy products, dietary fibre, whole grains, and protein from plant sources that is reduced in saturated fat and cholesterol (Nerenberg et al., 2018). Intervention studies implementing the DASH diet have been shown to produce reductions in both SBP and DBP (Moore et al., 2001; Sacks et al., 2001; Nerenberg et al., 2018). An accumulation of 60 to 150 minutes of aerobic exercise (intensity of 50-80% $VO2_{max}$) a week that can be completed in 10-minute minimum bouts, is recommended for the management of healthy BP (Nerenberg et al., 2018; Whelton et al., 2017). Furthermore, 2-3 days of dynamic resistance exercise a week at an intensity of 60-80% of a 1 repetition max (1RM) for 8-10 exercises (2-3 sets, 8-10 repetitions per set) is recommended as an adjunct to aerobic training (Nerenberg et al., 2018; Pescatello et al., 2015). Moreover, 4 × 2 min (hand grip), 1 minute of rest between exercises, 30%-40% maximum voluntary contraction (MVC) 3 times a week for 8-10 weeks is recommended in conjunction with aerobic exercise (Whelton et al., 2017). Unfortunately, if the lifestyle modifications listed above do not elicit adequate reductions in BP, pharmacotherapy is recommended to reduce BP to within the target clinical range (Whelton et al., 2017). Different classes of antihypertensive drugs can be implemented to control BP; commonly used medications are calcium channel blockers, angiotensin converting enzyme inhibitors, thiazide diuretics and beta-blockers (Whelton et al., 2017). Calcium channel blockers work by inhibiting calcium uptake into the vascular smooth muscle, blocking calcium influx (which, as previously mentioned, promotes dilation), lowering TPR and ultimately BP (GodFraind, 2006). Angiotensin converting enzyme inhibitors prevent the conversion of angiotensin I to the vasoconstrictor angiotensin II, thus decreasing TPR and BP (Kalyesubula et al., 2014). Thiazide diuretics prevent Na^+ reabsorption within the kidney (Duarte & Cooper-DeHoff, 2010). By reducing Na^+ reabsorption, fluid volume is reduced via increased

urine expulsion, thus resulting in a lowered Q, and ultimately a reduced BP (Duarte & Cooper-DeHoff, 2010). Although fluid volume is restored within 6 weeks of starting thiazide treatment, BP reductions are maintained, suggesting that chronic BP reductions are caused through a different mechanism or series of mechanisms, such as improved endothelium function or increased vasodilation, rather than solely fluid balance manipulation (Duarte & Cooper-DeHoff, 2010). Finally, beta-blockers inhibit sympathetic stimulation of the heart by preventing the binding of NE and E, resulting in reduced HR, Q, and BP (Ram & Venkata 2010).

Although the lifestyle modifications listed above have been shown to be effective in the management of BP, only 15% of Canadians are meeting the recommendations of daily physical activity (Statistics Canada, 2014a), and only half of Canadians are consuming the recommended servings of fruits and vegetables (Statistics Canada, 2012). Furthermore, 15% of hypertensive Canadians have "uncontrolled" HTN, meaning that, although they are optimally treated, these individuals still have resting BP values exceeding 140/90 mmHg (Statistics Canada, 2014b).

Adherence to these lifestyle modifications (i.e., exercise and diet) appears to be difficult for some individuals due to perceived barriers such as time, energy, and for those with lower socio-economic status. Options for safe physical activity (i.e., public recreational facilities, bike paths, usable sidewalks, parks and social support), as well as access to healthy food and safe drinking water, may be difficult (Middleton et al., 2013; Tarasuk et al., 2013). There is growing concern for true "resistant" hypertensive individuals, who, despite being prescribed 3 or more antihypertensive medications and being compliant with lifestyle modifications, still display BP measurements above clinically recommended ranges (Calhoun et al., 2008). Poor adherence to pharmacotherapy, due to avoidance of unwanted side effects, has also been reported as a potential reason for BP measurements not meeting clinical targets (Svensson et al., 2000). These

findings argue that current HTN interventions are not effective for everyone, and therefore, novel interventions must be explored.

1.3 Exercise Training

Exercise training plays an important role in the management and treatment of HTN. Currently, the Canadian Physical Activity Guidelines recommend 150 minutes of cumulated moderate to vigorous aerobic physical activity a week completed in ~10 minute bouts for individuals over the age of 18 years (Tremblay et al., 2011). Furthermore, 2 or more days a week of bone and muscle strengthening exercise focusing on major muscle groups has been recommended to add additional benefit (Tremblay et al., 2011). For hypertensive Canadians, 30-60 minutes of moderate-vigorous dynamic physical activity (i.e., walking, jogging, cycling or swimming) 4-7 days a week, in addition to their routine activities of daily living, is recommended as a means of reducing BP (Nerenberg et al., 2018). Dynamic resistance exercise (free weight lifting, fixed weight lifting and handgrip exercise) has been recommended as an adjunct to aerobic exercise (Nerenberg et al., 2018). Moreover, isometric resistance exercise (IRE), specifically isometric handgrip (IHG) exercise, has also been endorsed by the American Heart Association and the Canadian Hypertension Education Program as an alternative exercise intervention to lower BP (Whelton et al., 2017; Nerenberg et al., 2018). Isometric exercise involves a sustained contraction against an immovable load with little to no alteration to muscular length (Inder et al., 2016). The most widely used IHG exercise protocol involves 4, 2-minute isometric contractions at 30% of a maximum voluntary contraction (MVC) separated by a timed rest period, for a total duration of 12-15 minutes, completed at least 3 times a week for 8-10 weeks (Whelton et al., 2017). IHG exercise and its effects will be discussed in greater detail below (see Section 1.4 Isometric Handgrip Exercise).

1.3.1 Acute Effects of Aerobic Exercise on Blood Pressure

Following the initiation of a bout of aerobic exercise, SBP and HR are increased via reduced parasympathetic outflow and increased sympathetic outflow that increases Q 6-10 fold, and in turn increases BP (MacDonald, 2002). This elevation in BP is further exasperated by vasoconstriction within venous vasculature to increase venous return and SV (MacDonald, 2002). Furthermore, to match metabolic demand, blood flow is increased through vasodilation of arterioles that innervate more metabolically active tissues (i.e., skeletal muscle). In contrast, blood is shunted away from non-essential areas (i.e., digestive organs) via arteriole vasoconstriction. The increased vasodilation to working muscles serves as a buffer for the increase in BP achieved via augmented Q through reductions in TPR (MacDonald, 2002). The contracting muscles exert a substantial force onto the surrounding vasculature, and consequently, occlude blood flow (Delaney et al., 2010). As a result, metabolites such as K+, lactic acid, H+, and adenosine accumulate locally inside the exercising muscle tissue (Boushel, 2010). This increase in metabolic by-products leads to the activation of the exercise pressor reflex (EPR) (Delaney et al., 2010). The EPR sends afferent signals from the exercising muscles to the CC which evokes increased sympathetic activity that temporarily increases HR and BP in effort to restore perfusion of blood to working muscles (Delaney et al., 2010). The magnitude of the elevations in BP and HR are proportional to the length of time a muscle is contracted, the force at which the muscle is contracting, and the size of the active muscle (de Sousa et al., 2013; de Souza Nery et al., 2010; Rezk et al., 2006; MacDougall et al., 1985). Contraction of skeletal muscle also increases venous return of blood back to the heart and Q, resulting in increased BP (MacDonald, 2002). The decreased TPR noted above appears to have little effect on DBP during an acute bout of aerobic exercise, however, slight declines may occur due to blood flow increasing towards the periphery for heat dissipation (MacDonald, 2002).

Following an acute bout of aerobic exercise, both SBP and DBP can be reduced, a phenomenon known as post-exercise hypotension (PEH). Although PEH has been shown to occur in both hypertensive and normotensive individuals, this effect seems to be more prominent for those with higher BP (Cardoso et al., 2010). Reductions in BP can occur within a magnitude of 2-12 mmHg over the course of 4-16 hours in HTN individuals (Cardoso et al., 2010). Evidence suggests that these findings are less pronounced in normotensive individuals, reporting smaller reductions of approximately 12 mmHg SBP and 5 mmHg DBP (Pardono et al., 2015). These findings support the idea that PEH reductions in BP are proportional to BP prior to exercise, so that higher pre-exercising BP values appear to have greater reductions following exercise than lower pre-exercising values (Cardoso et al., 2010). The exact mechanism causing PEH remains unknown, but reductions in TPR due to localized vasodilatory substances, as well as decreased sympathetic outflow, have been implicated (Cardoso et al., 2010; MacDonald, 2002).

Acute aerobic exercise appears to have a lowering effect on ABPM in hypertensive populations (Ciolac et al., 2008; Cardoso et al., 2010). Following 40 minutes of cycling at an intensity of 60% heart rate reserve, participants experienced reductions in 24-hour ABPM of 3 mmHg SBP and 2 mmHg DBP (Ciolac et al., 2008). A meta-analysis has reported reductions as high as 12 mmHg SBP in hypertensive individuals (Cardoso et al., 2010). Furthermore, more evidence is required in order to determine whether these reductions are observed in normotensive individuals following acute aerobic exercise (Cardoso et al., 2010).

1.3.2 Chronic Effects of Aerobic Exercise on Blood Pressure

The BP lowering effects of chronic aerobic exercise have been well established (Cardoso et al., 2010). There is ample support for the efficacy of aerobic exercise intervention as an effective means of lowering resting BP (Pescatello et al., 2015). Despite differences in the

intensity of exercise (30-90% of maximum oxygen consumption), length of intervention (4-52 weeks), duration of exercise 1-60 minutes, frequency (1-7 days/week) and exercise modality (walking, running, swimming, cycling), most types of aerobic exercise seem to produce reductions in BP (Pescatello et al., 2015; Cornelissen & Smart, 2013; Cornelissen & Fagard, 2010; Kelley et al., 2001). Manipulation of these exercise variables (i.e., higher intensity with greater frequency) can produce more pronounced reductions in BP. (Pescatello et al., 2015; Cornelissen & Smart, 2013; Cornelissen & Fagard, 2010; Kelley et al., 2001).

The exact mechanisms responsible for the BP lowering effects of chronic aerobic exercise remain inconclusive. Due to the complex nature of HTN etiology, the mechanism is likely a result of different interactive BP regulatory (i.e., neural, hormonal and local) pathways. Current evidence suggests that reductions in BP following chronic aerobic exercise are attributed to decreases in TPR, as opposed to changes in Q (Fagard, 2006; Cardoso et al., 2010). Moreover, this reduction in TPR is likely attributed in part to reductions in SNS activity, which attenuates vasoconstriction (Fagard, 2006). Furthermore, increases in circulating NO and decreases in ET-1 induce greater dilation of blood vessels, resulting in lowered TPR and ultimately BP (McLean et al. 2015; Maeda et al., 2001).

The effects of aerobic training on ABPM requires further investigation. Chronic aerobic training appears to produce reductions in 24-hour ABPM of approximately 8 mmHg SBP and 4 mmHg DBP in normotensive, and 3 mmHg SBP and 4 mmHg DBP for hypertensive individuals, respectively (Cardoso et al., 2010). A recent meta-analysis examining the effects of $\geq$4 weeks of aerobic training has shown reductions in daytime ABPM of 3/3 mmHg in normotensives and 7/5 mmHg in hypertensive individuals' SBP and DBP, respectively (Cornelissen et al., 2013). However, aerobic training did not elicit any change in nighttime ABPM (Cornelissen et al.,

2013). The mechanism(s) behind these reductions remain equivocal, but likely reflect the local and neurological pathways that are responsible for the reductions in resting BP.

1.3.3 Acute Effects of Dynamic Resistance Exercise on Blood Pressure

An acute bout of dynamic resistance exercise, which involves utilizing muscular contractions of larger muscle groups to move a load placed upon that muscle (i.e., weight training), elicits an immediate and substantial increase in both SBP and DBP of upwards of >350/200 mmHg, respectively due to EPR (See Section 1.3.1; de Sousa et al., 2013; de Souza Nery et al., 2010; MacDougall et al., 1985). Individuals with HTN show greater elevations in SBP compared to normotensives when completing muscular contractions at matching intensities (de Souza Nery et al., 2010). These differences in BP response to dynamic resistance exercise are suggested to be the result of increased SNS activity at both rest and during exercise (de Souza Nery et al., 2010). Additionally, alterations in the RAAS system in hypertensive individuals may account for the greater BP attenuation (de Souza Nery et al., 2010).

With respect to PEH, an acute bout of dynamic resistance exercise can elicit reductions in resting BP and 24-hour ABPM (Cassonatto et al., 2016). A recent meta-analysis found that a single bout of dynamic resistance exercise reduces resting SBP by 3 mmHg and DBP by 3 mmHg at 1-hour post-exercise (Cassonatto et al., 2016). Greater reductions of 5/5 mmHg were observed at 90 minutes post-exercise for SBP and DBP, respectively (Cassonatto et al., 2016). Furthermore, a single bout of dynamic resistance exercise reduced 24-hour ABPM by 2/1 mmHg of SBP and DBP, respectively (Cassonatto et al., 2016). Notably, individuals with HTN observed greater reductions in resting BP (9 mmHg SBP and 5 mmHg DBP) compared to their normotensive counterparts (3 mmHg SBP and 3 mmHg DBP) (Cassonatto et al., 2016). Exercise intensity appears to influence PEH; resistance exercise completed at low intensity (40% of 1 repetition max (1RM)) reduced SBP by 6 mmHg, while high intensity (80% 1RM) reduced SBP

by 8 mmHg (Rezk et al., 2006). Interestingly, only the low intensity exercise appeared to have an effect on DBP (Rezk et al., 2006). The higher intensity resistance training (80% 1RM) elicited augmentation in TPR compared to the lower intensity resistance training (40% 1 RM). This increase in TPR is suggested to be a counter regulatory mechanism to combat decreases in Q caused by losses in SV because of blood being forced from the vasculature into the intracellular space (Rezk et al., 2006). Also, the increase in HR and BP that occur during resistance exercise is directly proportional to the intensity of exercise (Rezk et al., 2006; Lamotte et al., 2005). Although evidence supporting resistance exercise-induced PEH remains inconsistent, these reductions are believed to be caused by muscle mass reperfusion and are equivalent to the size of the muscle recruited, as well as the volume and intensity of the exercise performed (Cassonatto et al., 2016; Rezk et al., 2006; Lamotte et al., 2005; MacDougal, 2002; MacDougal et al.,1985).

1.3.4 Chronic Effects of Dynamic Resistance Exercise on Blood Pressure

The body of literature supporting the regulation of BP by chronic exposure to resistance exercise is not nearly as extensive as that of aerobic exercise training. A recent meta-analysis examining the effects of 14 weeks of moderate intensity dynamic resistance training on BP has shown reductions in normotensives and hypertensives of approximately 3/2 mmHg and 6/5 mmHg for SBP and DBP, respectively (MacDonald et al., 2016). Studies were matched for frequency (3 times per week), duration (14 weeks), intensity (60-70% 1RM) and training volume (3 sets, 11 repetitions) (MacDonald et al., 2016). As a result, the Canadian recommendations for hypertension management include dynamic resistance exercise as part of a comprehensive physical activity program to help reduce and maintain BP to within clinical target ranges (Nerenberg et al., 2018).

Despite the reductions in ABPM following acute dynamic resistance exercise noted above, there is still no evidence to suggest that there are chronic BP reductions elicited from

dynamic resistance exercise (Cardoso et al., 2010). This lack of evidence is the result of an insufficient number of studies examining the effects of chronic dynamic resistance exercise on ABPM (Cardoso et al., 2010). Therefore, more data are needed before a sufficient conclusion can be drawn with regards to the chronic effects of dynamic resistance training on ABPM.

1.4 Isometric Resistance Exercise (IRE)

1.4.1 Acute Effects of Isometric Resistance Exercise on Blood Pressure

IRE training is a novel BP-lowering exercise modality for the treatment of HTN. Typically, IRE protocols employ multiple, sustained muscular contractions at a set percentage of a maximum voluntary contraction (MVC), at which there are no changes to muscle length or joint angle (Millar et al., 2014). IRE has been recently endorsed by the American Heart Association and by Hypertension Canada as an alternative form of exercise for the treatment to decrease BP, particularly due to the ease of use and time-effective nature of this exercise modality (Whelton et al., 2017; Nerenberg et al., 2018; Carlson et al., 2014; Millar et al., 2014). The most common form of IRE is known as IHG training, which employs 4, 2-minute isometric contractions at an intensity of 30% of MVC value on a hand dynamometer, using alternate hands, conducted 3 times per week (Whelton et al., 2017).

The effects of IRE on PEH requires further investigation. In older normotensive individuals, reductions of 3 mmHg SBP have been observed immediately following 4, 2-minute IHG contractions at 30% MVC (Millar et al., 2009). In contrast, work by Ash and colleagues (2017) has shown that no PEH is observed in pre-hypertensives following the completion of 4, 2-minute IHG contractions at 30% MVC (Ash et al., 2017). Similarly, work from Bartol and colleagues (2012) did not observe PEH following IHG training in well-medicated hypertensives (Bartol et al., 2012). This lack of observable PEH could be attributed to the BP modulating

effects of anti-hypertensive medication (Millar et al. 2009). Further investigation is needed to understand the acute cardiovascular responses following an acute bout of IHG.

1.4.2 Chronic Effects of Isometric Resistance Exercise on Blood Pressure

The body of support for IRE training-induced reductions in BP is growing. Numerous studies have shown that IRE training can reduce resting BP in a variety of populations, using IHG and ILE training protocols (Inder et al., 2016; Carlson et al., 2014; Millar et al., 2014). In a recent meta-analysis, Inder and colleagues (2016) found reductions of 5 and 4 mmHg SBP and DBP, respectively, as well as a reduction in HR by 2 bpm following 8 weeks of IRE training in hypertensives and normotensives (Inder et al., 2016).

With respect to duration, participants who complete $\geq$8 weeks of IRE appear to have larger reductions in SBP (7 mmHg) compared to those who only completed <8 weeks of IRE (3 mmHg); duration did not appear to influence DBP (Inder et al., 2016). As far as frequency is concerned, work from Badrov and colleagues (2013) investigated the IHG dose-response in normotensive women (n=32; age 18-45 years). Participants completed either 3 (n=11 IHG training sessions per week, 5 IHG training sessions per week (n=12) or 0 training sessions per week (n=9) (4, 2-minute sustained contractions at 30% MVC) for 8 weeks. Although both groups produced reductions in SBP of approximately 6 mmHg, the 5 times per week group elicited these reductions earlier at 4 weeks, rather than at the end of 8 weeks for the 3 times per week group (Badrov et al., 2013).

The body of research surrounding the effects of IHG training on ABPM is scarce. Work from Somani and colleagues (2017) found significant reductions in day time, night time and 24-hour ABPM of 4 mmHg in young men and women with normal BP (Somani et al., 2017). Other work from Stiller-Moldovan and colleagues (2012) reported no significant change in medicated

hypertensives, although clinically relevant reductions were observed (Stiller-Moldovan et al., 2015). The lack of significant findings was suggested to be the result of pharmacotherapy proficiently controlling BP (Stiller-Moldovan et al., 2012). Further evidence is required to determine whether IHG training can effectively reduce ABPM.

Despite the growing evidence supporting IHG reductions in resting BP, the exact mechanism(s) responsible for these reduction remains unknown. IHG-induced reductions in BP obtained through IRE are suggested to occur through complex interactions of numerous mechanism(s) encompassing modulations of ANS activity and improvement in endothelium dependent vasodilation (McGowan et al., 2017; McGowan et al., 2006; McGowan et al., 2007a; McGowan et al., 2007b; Taylor et al., 2003; Millar et al., 2009; Badrov et al., 2013).

1.4.3 Surface Electromyography in Isometric Resistance Exercise

Surface EMG is a non-invasive technique used to measure the activity of muscle fibres. In brief, electrical activity of muscular contractions known as motor unit action potentials (MUAP) are tracked via electrodes placed on the surface of a muscle (Disselhorst-Klug et al., 2009). EMG data are comprised of the summation of MUAP, are representative of the magnitude and frequency of which a muscle is recruited, and are used as an indirect measure of muscle force (Farfán et al., 2010; Deluca, 1997). However, EMG does not directly measure force exerted from a particular muscle, but rather measures the recruitment of muscle based on the pattern of MUAPs (Deluca, 1997). Therefore, force can only be estimated and is subject to error during interpretation (Farfán et al., 2010; Deluca, 1997). A linear relationship has been shown between EMG activity and muscular force, meaning that higher EMG activity is correlated with higher forces exerted by the muscle (Disselhorst-Klug et al., 2009).

During an isometric contraction, where the length of the muscle remains constant, EMG activity can be reflective of forces exerted by the exercising muscle, as there is no change in joint angle or muscle length that can alter the data (Disselhorst-Klug et al., 2009; Deluca, 1997). Fatigue can influence EMG activity. As a muscle maintains a sustained contraction at a particular force, the muscle begins to fatigue (Deluca, 1997). As a result, muscles are recruited more frequently and with larger amplitudes to maintain the necessary force desired (Deluca, 1997; Solomonow et al., 1990).

A potential limitation of surface EMG is the error in data collection due to the electrodes picking up electrical activity from surrounding muscles in a phenomenon known as “cross-talk”, which can potentially mask the activity of the examined muscle (Disselhorst-Klug et al., 2009). Another limitation to EMG is that biological tissues like skin, adipose, and muscle conduct electrical signals differently and differences in body composition can interfere with the EMG electrodes picking up MUAP signals (Deluca, 1997). Furthermore, improper recording of EMG data can occur if electrodes do not have proper contact with the skin due to excessive hair, or oils from the skin (Deluca, 1997). Finally, errors can exist within the analysis technique of the investigator (Farfán et al., 2010). Analysis techniques utilizing the root mean square (RMS) of the EMG data have been shown to be reliable for the interpretation of force and fatigue during both dynamic and isometric muscular contractions (Fukuda et al., 2010; Farfán et al., 2010) Despite these limitations, surface EMG has been shown to be a reliable tool for indirect determination of muscle exertion (Diesselhorst-Klug et al., 2009).

Within the IRE literature, EMG is utilized as an alternative to MVCs as a determinant of exercise intensity (Deveraux et al., 2010; Wiles et al., 2010; Wiles et al., 2008). Participants perform maximum voluntary contractions, from which their peak EMG activity were obtained

(Wiles et al., 2008). The exercise intensity was incrementally increased from 10-30 % peak EMG activity, during which HR and BP were recorded (Wiles et al., 2008). EMG activity was positively correlated with increases in both HR and SBP (Wiles et al., 2008). Other work from Wiles and colleagues have utilized EMG to match exercise intensity to HR in order to produce increases in SBP and HR similar to those utilizing MVCs (Wiles et al., 2010). Alternatively, the literature surrounding EMG activity during a bout of IHG exercise is scarce. Some studies have examined the effects of EMG and BP in response to IHG exercise (Jacobsen et al., 1994; Cotzias & Marshall, 1993). It is interesting to note that higher EMG activity in the non-exercising limb during the IHG exercise was found to be associated with decreased TPR during the contraction (Jacobsen et al., 1994; Cotzias & Marshall, 1993). Despite these reductions in TPR elicited from IHG, the relationship between EMG in the exercising arm and BP in response to IHG exercise, remains to be quantified.

1.4.4 Feasibility of Isometric Resistance Exercise Devices

As noted above, isometric exercise has been shown to be an effective modulator of resting BP. Although IRE training has been shown to elicit reductions in BP, protocols have primarily been contained to a laboratory setting due to the equipment used (see below for details). Although recent evidence (Goldring et al., 2014; Wiles et al., 2016) suggests that home-based IRE training may be feasible, due to the portable nature of IHG devices and the use of hands versus legs (minimizes influence of lower body mobility and/or joint-related issues), IHG training may be a more favorable exercise modality. Unfortunately, 85% of Canadians are not meeting daily recommended exercise requirements, and with a growing number of Canadians who are unable to control their BP, novel exercise strategies must be explored (Statistics Canada, 2014a; Statistics Canada, 2014b). Therefore, the implementation of IHG training may be one such novel intervention that can be easily implemented into daily living and may promote better

exercise adherence. Currently, the computerized dynamometers utilized in many IHG training studies to date cost upwards of $500 CAD (Zona Health, 2018). The cost associated with purchasing IHG equipment potentially establishes an economic barrier for those with HTN who cannot afford this alternative exercise modality.

Currently, studies examining the effects of cost-effective alternatives to traditional IRE devices are limited and involve small numbers of primarily normotensive participants. Garg and colleagues (2014) examined the effects of 10 weeks of IHG training on resting BP in young normotensives utilizing a spring-loaded handgrip (Garg et al., 2014). Participants completed 5, 3-minute sustained contractions at 30% MVC, 3 times a week for 10 weeks (Garg et al., 2014). Upon completion of the 10 weeks of training, resting BP was reduced 10 mmHg and 6 mmHg SBP and DBP, respectively (Garg et al., 2014). This is consistent with the findings from Millar and colleagues (2008) who examined the effects of IHG training performed 3 times a week for 8 weeks utilizing an inexpensive spring-loaded handgrip device in older normotensive individuals (Millar et al., 2008). Participants were paired with a spring-loaded handgrip device with a low, medium or high resistance, respectively, which corresponded to approximately 30-40% MVC calculated utilizing a computerized hand dynamometer (Millar et al., 2008). Following 8 weeks of IHG training, significant reductions in resting BP of 10 and 3 mmHg SBP and DBP, respectively were obtained (Millar et al., 2008). These findings suggest that similar reductions in resting BP can be achieved utilizing less expensive alternative IHG devices. However, the handgrips had to be outfitted with implanted pressure gauges by the research investigators, and thus remain problematic for large-scale prescription. Furthermore, little is understood about the acute cardiovascular or neuromuscular responses of these IHG alternative devices and no study to date has directly compared devices.

In addition, EMG equipment is expensive and requires training for proper use, making it an unfeasible exercise modality for the average person (Wiles et al., 2016). This gap in easily available home IRE exercise sparked a study by Goldring and colleagues in 2014 that attempted to quantify a stimulus using a static wall sit exercise, similar to traditional IRE training, that could elicit reductions in BP (Goldring et al., 2014). Normotensive participants completed 2 minutes of isometric wall squats with knee angles from 135 to 90 degrees in 5-degree intervals, with BP and HR measured at rest and continuously throughout the 2-minute contractions (Goldring et al., 2014). An inverse relationship was discovered between knee angle and BP and HR (Goldring et al., 2014). This allows for incremental determination of exercise intensity that can match traditional IRE literature utilizing an intensity of 95% peak HR (Goldring et al., 2014; Deveraux et al., 2010; Wiles et al., 2010). A study conducted by Wiles and colleagues (2016), also with young, healthy normotensives, examined the effects of utilizing isometric wall sits on resting BP in a training study (Wiles et al., 2016). Participants completed 4, sustained 2-minute sits at a participant-specific knee joint angle that corresponded to 95% peak HR, 3 times a week for 4 weeks (Wiles et al., 2016). Following 4 weeks, resting BP was reduced by 4 and 3 mmHg SBP and DBP, respectively, as well as reduced resting HR by 5 beats per minute (Wiles et al., 2016).

These results support the use of IRE as an effective means to lower resting BP that can be completed without the use of expensive equipment in the comfort of one's own home. Further research is required to support the real-world implementation of IRE and help to reveal the potential mechanism(s) of action regarding how IRE lowers resting BP.

1.4.5 Gaps in the Existing Isometric Resistance Exercise Literature

The body of support for IRE training as a means of lowering resting and ambulatory BP is continually growing. Despite the recent endorsements from the AHA and Hypertension

Canada, IRE has yet to be as widely accepted as traditional aerobic and dynamic resistance exercise as an effective exercise modality (Whelton et al., 2017; Nerenberg et al., 2018). As noted in Section 1.4.3 above, cost-effective alternatives appear to induce similar reductions in resting BP as traditional IRE equipment (Wiles et al., 2016; Garg et al., 2014; Goldring et al., 2010; Miller et al., 2008). Despite these similarities, the feasibility of matching the exercise intensity between devices, and quantifying the acute effects of IHG exercise utilizing inexpensive IHG devices, remains to be explored.

Because the exact mechanism(s) of how IRE-reduced BP have yet to be fully determined, it is still valid to examine responses to different exercise stimuli. Currently, evidence directly examining the differences in BP, HR, or muscular activation between traditional IRE devices and cost-effective alternatives, is limited. The findings from Goldring et al. (2010) and Wiles et al. (2016) indicate that quantifying the exercise stimulus allows for matching of exercise intensity without the need for expensive equipment that can elicit reductions in resting BP and HR (Goldring et al., 2010; Wiles et al., 2016).

During sustained muscular contractions, subjective ratings of perceived exertion (RPE) have been correlated with muscular activation (via EMG) and have been shown to be predictive of fatigue (Troianoa et al., 2008). However, little is known of the perceived effort and discomfort associated with IRE exercise itself and how effort, pain or fatigue may affect implementation of IRE as a mainstream exercise tool. Work from Wiles and colleagues (2005) examined the BP, HR and RPE between three IRE devices; one IL device utilizing a force transducer, two utilizing an arm contraction at the elbow (one utilizing a novel device and one utilizing a force transducer) (Wiles et al., 2005). Participants completed 4, 2-minute sustained contractions on each device while SBP, DBP, HR and RPE were collected throughout (Wiles et al., 2005). Significant

differences were found only in SBP between the two upper limb devices with the transducer device eliciting an elevation of 9 mmHg greater than the novel device (Wiles et al., 2005). No significant differences were found for DBP, HR or RPE between the three devices (Wiles et al., 2005). Integration of both subjective and objective measures of physiological response to IRE may provide insight into not only the effectiveness of inexpensive IRE equipment, but also the feasibility of implementing less expensive IRE devices to lower BP.

Despite the effectiveness of IHG exercise at reducing BP, it has only recently made it into international guidelines (Whelton et al., 2017; Nerenberg et al., 2018). Furthermore, more evidence is required into cost-effective alternatives. Furthermore, the acute IHG stimulus for these inexpensive alternatives remains to be investigated. Moreover, whether alternative devices are comparable in terms of subjective ratings of exertion may provide insight into how adherence to exercise training interventions would be for these handgrips. Additionally, understanding the acute stimulus may provide insight into the potential mechanism(s) responsible for IHG-induced BP reductions.

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Chapter 2: Research Project

2.1 Introduction

Hypertension (HTN), or high blood pressure (BP), is an increasing public health concern. Affecting nearly 5.3 million Canadians (Statistics Canada, 2013), hypertension increases an individual's chances of developing cardiovascular diseases (CVD), such as a heart attack or stroke (Public Health Agency of Canada, 2009). Identified recently as a global health crisis by the World Health Organization (WHO), hypertension is defined as a chronic elevation in resting BP of $\geq$130/80 mmHg or 135/85 mmHg using automated office BP (AOBP) measurements (Nerenberg et al., 2018; Whelton et al., 2017). Lifestyle modifications such as cessation of smoking, limiting alcohol consumption, proper nutrition and participation in regular exercise are cornerstone treatments for BP management (Nerenberg et al., 2018; Whelton et al., 2017; Blumenthal et al., 2010). In many cases, pharmacotherapy is prescribed in addition to traditional lifestyle modifications to reach target BP values (Nerenberg et al., 2018). Upwards of 50% of people treated for hypertension in Canada are not controlled to target levels (Nerenberg et al., 2018). Failure to achieve target BP greatly increases the risk of HTN-related complications such as a heart attack or stroke (Sarafidis, 2011). Thus, the implementation of new treatments that effectively lower, and then maintain BP, is essential.

Isometric handgrip (IHG) training is one such novel intervention which has been put forth by international organizations as an effective intervention to achieve BP control (Nerenberg et al., 2018; Whelton et al., 2017). IHG training, which employs 4, 2-minute sustained contractions at an intensity of 30% of maximum voluntary contraction (MVC) on a hand dynamometer, using alternate hands, is conducted 3 times per week (Whelton et al., 2017).

Numerous studies have shown that this simple and time-efficient form of exercise training can lower BP (i.e., mean reductions between 3 to 6 mmHg systolic BP and 4-6 mmHg diastolic BP) across a wide array of populations ranging from young normotensive individuals to

older individuals with diagnosed hypertension (McGowan et al. 2017; Inder et al., 2016; Carlson et al., 2014; Miller et al., 2014).

A current barrier to the implementation of IHG training as a mainstream intervention tool for those with hypertension, and thus the widespread endorsement of the American Heart Association/Canadian Hypertension guidelines in clinical practice and community-based exercise programs, is the high cost associated with the traditional computerized IHG dynamometers. Currently, the price for these traditional computerized devices cost upwards of $500 (CAD) (Zona Health, 2018). Although there is some research to suggest that less expensive spring-loaded devices offer similar benefits to more costly alternatives, the exercise in this work was limited to a laboratory setting (Garg et al., 2014; Millar et al., 2008). More specifically, the devices required research investigators to outfit the handgrips with pressure gauges to calculate appropriate exercise intensity, and thus remain problematic for large-scale prescription within the community (Garg et al., 2014; Millar et al., 2008). Thus, research into the effectiveness of alternative, commercially available, inexpensive handgrips is necessary.

As a preliminary step before implementing training studies utilizing cost-effective IHG devices, the IHG stimulus must be quantified in attempt to best match the response from existing, and effective, computerized IHG device. By better understanding what happens to the cardiovascular and neuromuscular systems during an acute bout of IHG utilizing the traditional and less costly IHG devices, a better understanding of the associated BP-lowering mechanism(s) may result. No study to date has attempted to compare the IHG stimulus across devices. Moreover, other IRE studies have used surface electromyography (EMG) in order to determine exercise intensity by measuring muscular activation (Wiles et al., 2010). Therefore, by muscle recruitment via surface EMG that occurs as a result of using the traditional computerized

dynamometer, to that from an inexpensive mechanical handgrip device, may allow for a more accurate comparison of devices. Additionally, there is little known about how a bout of IHG is perceived by the participant (e.g., how much effort is involved in performing the exercise and/or any associated discomfort). Integrating psychophysiological measures such as ratings of perceived exertion (RPE) and subjective discomfort through a numerical pain rating scale for pain (NRS Pain) will help determine whether alternative devices are comparable to traditional devices in terms of perceived effort and discomfort. Furthermore, developing a better understanding of the psychophysical factors associated with IHG training may provide insight into the potential adherence of IHG as a mainstream exercise tool to lower BP.

2.2 Research Questions

Research Question 1: Will an acute bout of IHG utilizing an inexpensive mechanical handgrip device elicit a similar cardiovascular response (HR and BP) as the traditional computerized dynamometer?

Research Question 2: Will an acute bout of IHG utilizing an inexpensive mechanical handgrip device elicit similar muscular activation as the traditional computerized dynamometer?

Research Question 3: Will an acute bout of IHG utilizing an inexpensive mechanical handgrip device elicit similar subjective ratings of perceived exertion (RPE) and pain on a numeric rating scale (NRS Pain) during IHG exercise?

2.3 General Methodology

2.3.1 Study Participants

Thirty-one young (age=18-30 years), healthy [no overt disease, not taking prescription medication with the exception of birth control pills (women were tested in the early follicular phase of the menstrual cycle if not on hormonal contraceptives, or during the low hormone phase

if on contraception) and free of physical limitations to use the devices] participants were recruited from the University of Windsor population and surrounding area. This included undergraduate and graduate students, faculty and staff, and community members. With an assigned alpha of 0.05, and a beta of 0.8, sample sizes ranging from 36 to 40 were estimated to effectively examine the electrical activity of the muscles of the upper extremity, cardiovascular responses, and subjective ratings to a single bout of IHG (Faul et al., 2007). Potential participants were recruited via poster campaign, presentations to research laboratories, undergraduate classes and/or community exercise facilities (e.g., gyms, community centres), word of mouth, and/or via email (See Appendix A for recruitment materials). Participants were excluded from engaging in the study if resting AOBP exceeded 135 and/or 85 mmHg SBP and DBP, respectively. Participants were also excluded if there was the presence of overt disease; if they were taking prescription medication (except for contraceptive medication); if there was the presence of physical limitations that impeded the use of the training devices; and/or if the participant indicated an allergy to any of the materials used for attaching the EMG instrumentation to their skin (e.g., electrode adhesive, adhesive on tape).

2.3.2 Study Design

Overview:

Eligible participants performed a bout of IHG on a computerized IHG dynamometer (IBX H-101, Zona Health, Boise, ID, USA; See Appendix B) and an inexpensive, store-bought mechanical grip (239247305, www.dhgate.com; See Appendix C) during a single testing session. A 30-minute stabilization period separated each bout. During each bout, beat-to-beat HR, minute-to-minute BP, ratings of perceived exertion, pain ratings and unilateral EMG data of the upper extremity were obtained. The total time to complete the collection procedures was approximately 4 hours, which included 3 points of contact, as described below.

Eligibility and Familiarization Session:
Visit 1 (Figure 1. A)

First, the primary investigator thoroughly explained the study, and asked potential participants to read a consent form and information sheet related to the study (See Appendix D and E). Those still interested in taking part in the study signed a consent form and were given a copy of the information sheet for their personal records.

The participants who provided consent completed a medical questionnaire and a Physical Activity Readiness Questionnaire (See Appendix F), with the intent of screening for the presence of any condition that would exclude them from the study. As the last step to determine eligibility, potential participants had their resting AOBP measured in their dominant arm to ensure they met the inclusion criteria (<135/85 mmHg). Resting AOBP was measured per standard laboratory protocol. In brief, resting AOBP was measured following 10-minutes of seated rest using brachial artery oscillometry (Dinamap Carescape v100, Critikon, Tampa, Florida, USA; See Appendix G). AOBP was measured by placing a cuff on the upper portion of the participant's dominant arm, and the cuff was automatically inflated to a pressure greater than the systolic BP to occlude the brachial artery. Four measurements were acquired, with 2-minute rest periods between each measurement, and the last 3 BP values were averaged. Using the BP data, together with the medical questionnaire, final eligibility of the potential participant was determined. At this time, participants were encouraged to ask any remaining questions, and were reminded of their right to withdraw from the study at any time. Eligible participants were then scheduled for a familiarization session, where they had an opportunity to practice all portions of the investigation.

Familiarization Session

Visit 2 (Figure 1.B)

At least 24 hours after the initial visit, a familiarization session occurred. Interested participants who met the inclusion criteria, were still interested in engaging in the study, and who refrained from vigorous physical activity over the previous 24 hours, were tested 2 hours post-prandial and at least 12 hours post-caffeine. To minimize the effects of a full bladder on BP as having a full bladder can raise BP, participants were asked to void prior to the familiarization session.

Participants provided on-going consent by initialling the visit 2 consent section of the consent form. They were then given 10 minutes of seated rest after which 4 resting BP values were taken as noted above. Following those measures, they were outfitted with the necessary equipment to gather EMG data. More specifically, two electrodes were attached to the largest part of each muscle, parallel to the primary fascicle direction of the following muscles: flexor carpi ulnaris (FCU), brachioradialis (BR), extensor carpi ulnaris (ECU), brevis/longus, biceps brachii (BB) and triceps brachii (TB) with the arm held at a 90 degree angle at the elbow. The placement for the electrodes were as follows: BB: 1/3 of the distance proximally from the cubital fossa between the acromion and cubital fossa. BR: 2 finger breadths from the cubital crease with forearm in neutral position. TB: 2 finger breadths medially at 50% of distance between the acromion and the olecranon. ECU: 1/3 of the distance from the lateral epicondyle between the lateral epicondyle and the ulnar styloid. FCU: 2 fingerbreadths from the ulnar border on the proximal third of the forearm (Zip, 1982) (Figure 2). The skin to which the electrodes were attached was shaved with a disposable safety razor (if needed) and then cleaned with an alcohol swab prior to attaching the electrodes (PUN-96, Bortec Biological Limited, Calgary, Alberta,

Canada; See Appendix H). A low allergy surgical tape was placed over the electrodes and adhered to the skin to provide additional adherence and limit any electrode movement during data collection.

Participants were then randomly assigned to either a computerized IHG dynamometer (IBX H-101, Zona Health, Boise, ID, USA); Appendix B) or an inexpensive store-bought mechanical grip (> $10) (239247305, www.dhgate.com; Appendix C). They were then asked to perform 3 maximal 2-second squeezes separated by 1-minute of rest between squeezes. Following the third squeeze, participants completed one 2-minute sustained contraction at an intensity of 30% of their maximum squeeze, during which BP and HR measurements were taken each minute. Following the completion of each 2-minute contraction, the participant was asked to rate their perceived exertion using the Borg 6-20 rating of perceived exertion scale (Appendix I; Borg, 1998) and to rank any discomfort that they may have experienced on a 1-10 numeric pain scale (Appendix J; Borg, 1998). After a 10-minute rest period, participants completed the same protocol utilizing the other handgrip device. Once the aforementioned protocol was completed, and if the participant was still interested in engaging in the study, visit 3 was scheduled at least 24 hours later.

Testing Day

Visit 3 (Figure 1C)

At least 24 hours after visit 2, a single testing session occurred. All participants were tested in the morning in a temperature-controlled room (PACR Laboratory). Participants were asked to have refrained from vigorous physical activity over the previous 24 hours, were tested 2 hours post-prandial and at least 12 hours post-caffeine. To minimize the effects of a full bladder

on BP, participants were asked to void prior to testing. Participants were asked to provide on-going consent by initialling the visit 3 section of the consent form.

Participants were seated for the duration of the testing period, with both their right and left forearms resting on a table in front of them with their elbows at approximately 90 degrees. First, participants were outfitted with the necessary equipment to acquire BP and HR (See Appendix G), and EMG (See Appendix H). Resting AOBP was measured following 10 minutes of seated rest, as described above. Next, participants performed (in random order) an IHG protocol on the traditional, computerized IHG dynamometer and the mechanical grip. The protocols were separated by at least 30-minutes of rest or until the participant's BP returned to near pre-exercise values. In both conditions, the IHG bout consisted of 4, 2-minute unilateral (non-dominant arm) contractions at 30% of their MVC, each separated by a 4-minute rest period. The procedure for determining exercise intensity was as follows: 1) For the computerized dynamometer, MVC was determined at the onset of the protocol in the non-dominant hand using linear load cells inherent to the device, whereby 30% MVC was automatically calculated by the device using these data, 2) For the mechanical handgrip, the MVC was determined by having the participant maximally squeeze the handgrip, again with their non-dominant hand, against the resistance of the handgrip which was quantified by turning a dial on the device. Therefore, 30% MVC was manually calculated based on the maximum load determined from the dial. Upon completion of each 2-minute contraction, the participant was asked to rate their perceived exertion using the Borg 6-20 rating of perceived exertion scale (RPE) (See Appendix I; Borg, 1998), and to rank any discomfort that they may have experienced on a 1-10 numeric rating scale for pain (NRS Pain) (See Appendix J; Hawker et al., 2011).

All data were collected continuously for 10 minutes prior to, during, and for at least 30 minutes following each IHG bout. Specifically, AOBP was measured each minute in the dominant (non-exercising) limb using the device described above. During each bout of the 4, 2-minute contractions, the peak value of SBP, DBP and HR for both the mechanical and computerized IHG devices were obtained every minute utilizing automated oscillometry. Next, pre-exercise values (blood pressure reading taken just before the onset of the exercise) were subtracted from peak exercise values for each of these three variables in order to create a delta score for each variable. These delta scores were then compared between the two devices to determine the peak acute stimulus for SBP, DBP and HR. On the non-dominant (exercising) limb, EMG data were obtained using the same protocol described above**.** During each 2-minute contraction, raw surface EMG data were collected at a sampling rate of 2k/s. Raw EMG data were rectified, filtered using a low pass Butterworth filter with a cut-off frequency of 2 Hz, and normalized to the peak activity of 2-second MVC trials. All data was normalized to each participant peak surface EMG score from the 2-second contraction for each muscle. Normalized EMG was averaged over time intervals (2 sec) representing 5 epochs during each contraction. Participants were encouraged to keep their arm flexed at a 90-degree angle throughout the 2-minute contractions to prevent changes in joint angle and muscle length from influencing the EMG data. Surface EMG data were subdivided into 5, 2-second time epochs within 22 second time intervals spanning 110 seconds (first 10 seconds of each contraction was excluded to accommodate the participant acclimating to 30% MVC resistance) (Figure 3). Upon completion of each 2-minute contraction, participants were asked to rate their RPE (See Appendix J) and NRS-Pain (See Appendix K) during the isometric exercise.

(A)

Inclusion and exclusion criteria

-Reviewed and signed consent form
-Receive letter of information
-Medical questionnaire and PAR-Q

**Resting BP ≤ 135/85mmHg: schedule visit 2

-10 min. seated rest
-Resting BP and HR measured (4X, 2 min. rests between measurements)

(B) **Familiarization Day (~ 1 hr.)**

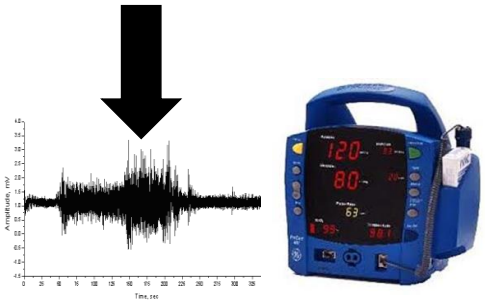
- No alcohol for 24 hours
- No vigorous exercise for 24 hours
- No caffeine for 12 hours
- 2 hours post-prandial
- Refrain from lotion
- Empty bladder
- Isolated, quiet, temperature-controlled room

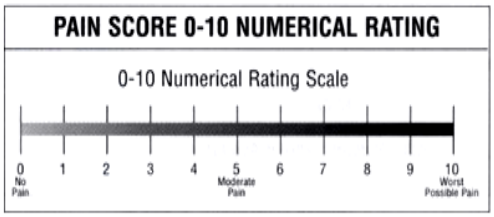
10 min. seated rest

4 resting BP/HR measurements, 2 min. rest intervals in between

Random assignment of 3, 2-sec. MVC with 1-min rest intervals in between. Followed by 1, 2-minute unilateral IHG contraction at 30% of the last MVC

10 min. stabilization between trials





Rating	Perceived Exertion
6	No exertion
7	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion

(C) **Testing Day (~ 2.5 hr.)**

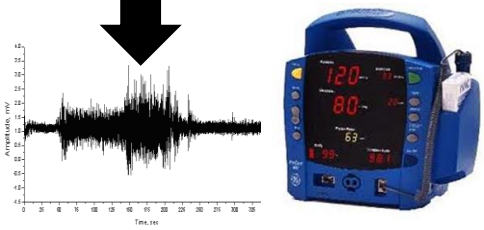
(A) No alcohol for 24 hours
(B) No vigorous exercise for 24 hours
(C) No caffeine for 12 hours
(D) 2 hours post-prandial
(E) Refrain from lotion
(F) Empty bladder
(G) Isolated, quiet, temperature-controlled room

10 min. seated rest

Random assignment of 4, 2-min unilateral IHG contractions at 30% MVC with 4 min. rest intervals in between. 30 minute stabilization between trials

Random assignment of 3, 2-sec. MVC with 1-min rest intervals in between. Followed by 1, 2-minute unilateral IHG contraction at 30% of the last MVC

10 min. stabilization between trials



Rating	Perceived Exertion
6	No exertion
7	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion

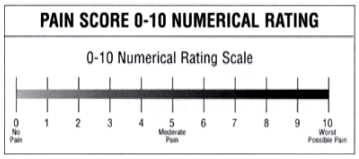


Figure 1: Study design: (A) Visit 1: initial visit: determination of eligibility is conducted (B) Visit 2: familiarization session where participants experience all testing procedures and protocol prior to testing (C) Visit 3: testing day

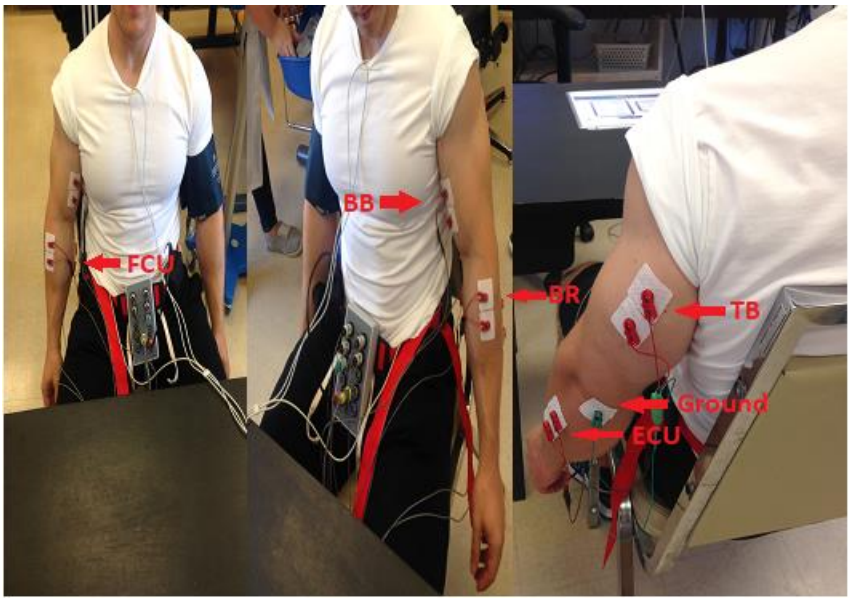


Figure 2: EMG Electrode Placement of electrodes on the non-dominant arm.

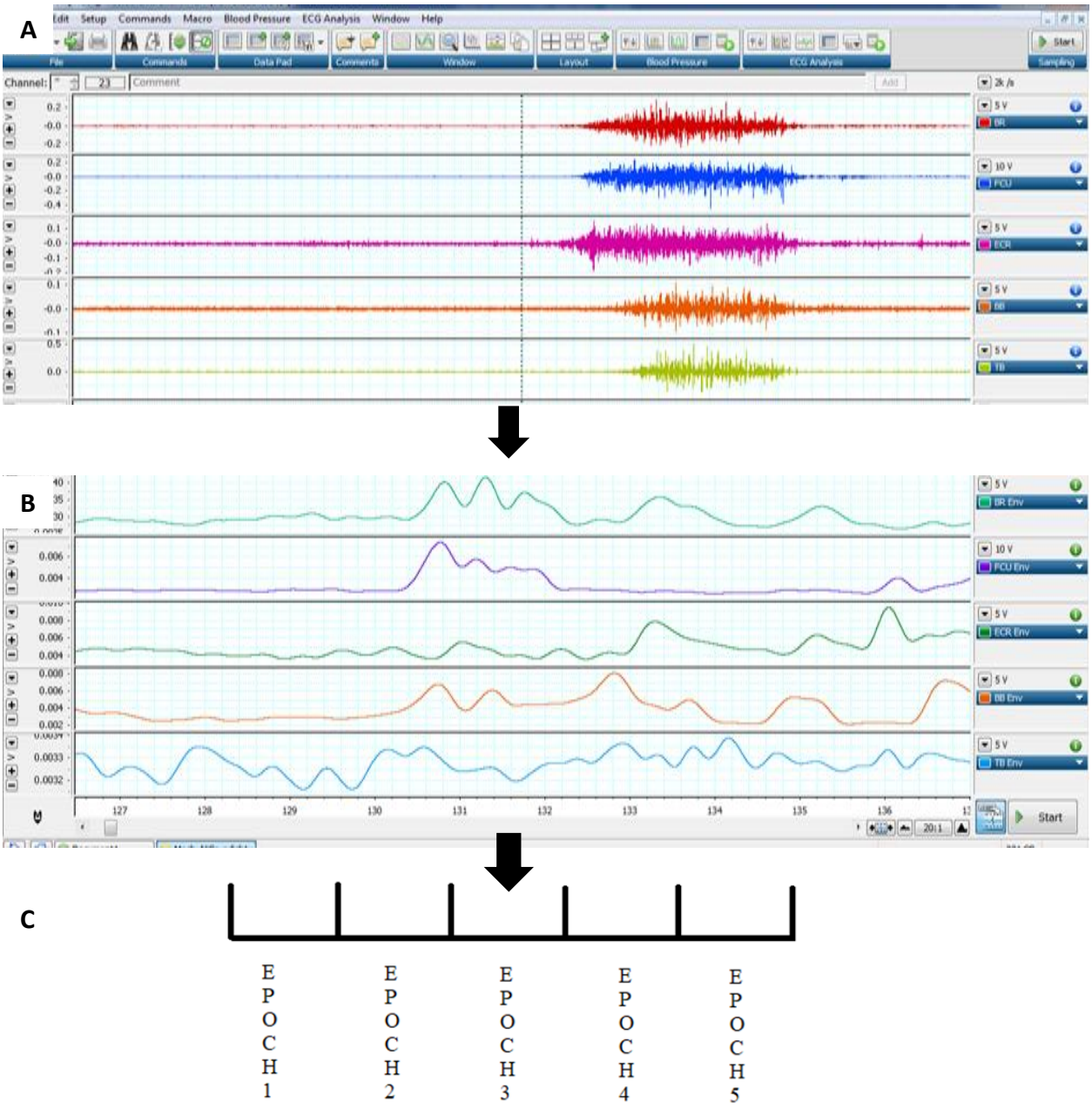


Figure 3: EMG data analysis protocol for extracting surface EMG data and binning of the data for analysis. Raw EMG was collected at a sampling rate of 2k/s (A). Data was rectified and a Butterworth filter with a cut-off frequency of 2 Hz was added to provide a linear envelope (B). Linear envelope data corresponding to 2- second MVC trials and 2-minute contractions were used from each muscle for analysis. Peak EMG scores for each muscle during the MVC trial were used to normalize data. The first 10 seconds of each 2-minute contraction was excluded to allow for acclimatization to the 30% MVC exercise intensity. The remaining 110 seconds were subdivided into 5, equal 22-second intervals. A 2-second epoch was taken from the middle of each of the 5 time intervals and used for analysis (C).

2.3.3 Statistical Analysis

To examine the acute exercise effects of the independent variable of IHG device type (computerized and mechanical) on the dependent variable of peak change in arterial BP (SBP and DBP) and HR across the 4 isometric contractions, a 2 x 4 repeated measures ANOVAs was conducted. Furthermore, the dependent variable of muscular activation (i.e., EMG) of the exercising arm for the BR, FCU, ECU, BB and TB muscles was analysed using a 2 x 4 (IHG type x contraction repeated measures ANOVA. Finally, both subjective measures of exertion (RPE and NRS Pain) with the device type as independent variable and peak rating score as dependent variable were analyzed utilizing a 2 x 4 (device type x peak rating score per contraction) repeated measures ANOVA. Sex (male or female), and order (starting handgrip) were analyzed as confounding variables to rule out the potential of these factors influencing the dependent variables. All data were analyzed using IBM SPSS Statistics 21 software (SPSS Inc., Chicago, Illinois, USA), data are presented as means and SD unless otherwise noted, and statistical significance was determined at $p < 0.05$.

2.4 Results

Of the 33 participants recruited, 5 were excluded based on not meeting the eligibility criteria (i.e. resting BP above 135/85 mmHg, the presence of a diagnosed medical condition or taking prescription medication other than birth control medication). Additionally, at visit 1, 2 eligible participants withdrew from the study for personal reasons. Furthermore, 6 participants withdrew from the study following visit 2 and did not complete visit 3, and therefore were excluded from data analyses. Twenty participants completed all three visits of the study and were included for analysis. Mean participant characteristics are outlined in Table 1.

Table 1: Participant characteristics

Characteristics	
N	20
Women	9
Age (years)	22.4 ± 2.2
Mass (kg)	77.8 ± 27.0
Height (cm)	163.4 ± 18.3
Resting SBP (mmHg)	113 ± 11
Resting DBP (mmHg)	63 ± 6
Resting MAP (mmHg)	80 ± 7
Resting HR (bpm)	70 ± 8

SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate. Values are mean ± SD.

2.4.1 Comparison of Acute Effects of Isometric Handgrip Exercise on Blood Pressure and Heart Rate Between Handgrip Devices

No statistical differences were observed for peak SBP ($p = 0.11$) and HR ($p = 0.18$) between devices (Table 2). Moreover, a statistically significant interaction was found between device type and sex for SBP ($p = 0.05$). A Tukey's post hoc test revealed that a statistically significant elevation in SBP for male participants using the mechanical handgrip (15 ± 11 mmHg) compared to the computerized device (7 ± 14 mmHg) ($p = 0.00$). A statistically significant increase was observed in DBP ($p = 0.03$) using the computerized device mmHg compared to the mechanical handgrip. Additionally, a significant interaction was found for device type and sex for DBP ($p = 0.02$). A Tukey's post hoc analysis revealed that when using the mechanical device, male participants had higher DBP elevations than did their female counterparts. Furthermore, no significant difference was found for potential confounding

variables of contraction or device starting order (all p>0.05). However, due to the low observed power across measures, these results should be interpreted with caution.

Table 2: Acute cardiovascular effects of isometric handgrip exercise utilizing the mechanical and computerized devices

Change from Pre-Exercise to Peak Exercise Values	Mechanical Handgrip Device	Computerized Handgrip Device	Observed Power
SBP (mmHg)	12 ± 13	8 ± 10	0.35
DBP (mmHg)	5 ± 13	6 ± 10 *	0.63
HR (bpm)	1 ± 9	9 ± 8	0.26

SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate. Values are mean ± SD. *Significant difference between handgrip devices for DBP p <0.05.

2.4.2 Comparison of Acute Effects of Isometric Handgrip Exercise on Muscular Activation Between Handgrip Devices

A statistically significant interaction was observed for the BB (p = 0.03) and BR (p = 0.03) for device and contraction. However, for the purposes of this study these were deemed non-meaningful relationships and therefore main effects were examined. for all the muscles. No statistically significant interactions were found between the two devices for the BR, FCU or TB muscles (all p > 0.05). However, statistically greater elevations in muscle activation were observed when using the mechanical versus computerized device for BB (p = 0.02) and ECU (p=0.00) (Table 3). Due to the low observed power, these findings should be interpreted with caution.

Table 3: Acute muscle activations (%MVC) of isometric handgrip exercise utilizing the mechanical and computerized devices

Change from %MVC	Mechanical Handgrip Device	Computerized Handgrip Device	Observed Power
BR	321.5 ± 322.9	304.7 ± 318.7	0.46
ECU	183.6* ± 130.0	173.6 ± 124.5	0.83
FCU	432.9 ± 905.3	402.1 ± 822.6	0.25
BB	175.6* ± 247.3	163.8 ± 235.1	0.67
TB	254.8 ± 201.5	238.1 ± 204.6	0.13

BR, brachioradialis; FCU, flexor carpi ulnaris; ECU, extensor carpi ulnaris; BB, biceps brachii and TB, triceps brachii Values are mean ± SD. *Significant difference BB and ECU ($p<0.05$).

2.4.3 Comparison of Acute Effects of Isometric Handgrip Exercise on Subjective Ratings of Perceived Exertion and Pain Between Handgrip Devices

No statistically significant differences were observed in either RPE ($p = 0.12$) or NRS-Pain scores ($p = 0.57$) between devices (Table 4). A statistically significant main effect for contraction on RPE scores was observed ($p = 0.00$). A Tukey's post hoc test revealed that with both devices, contractions 3 and 4 yielded elevations in RPE scores as compared to contraction 1. However, a statistically significant interaction was found for sex and contraction ($p =0.01$) for RPE. Furthermore, no statistically significant differences were found for the potentially confounding variables of device starting order for both RPE and NRS-Pain scores. Interestingly, when participants were asked to state which handgrip was their preferred device, all 20 participants stated that they preferred the computerized handgrip.

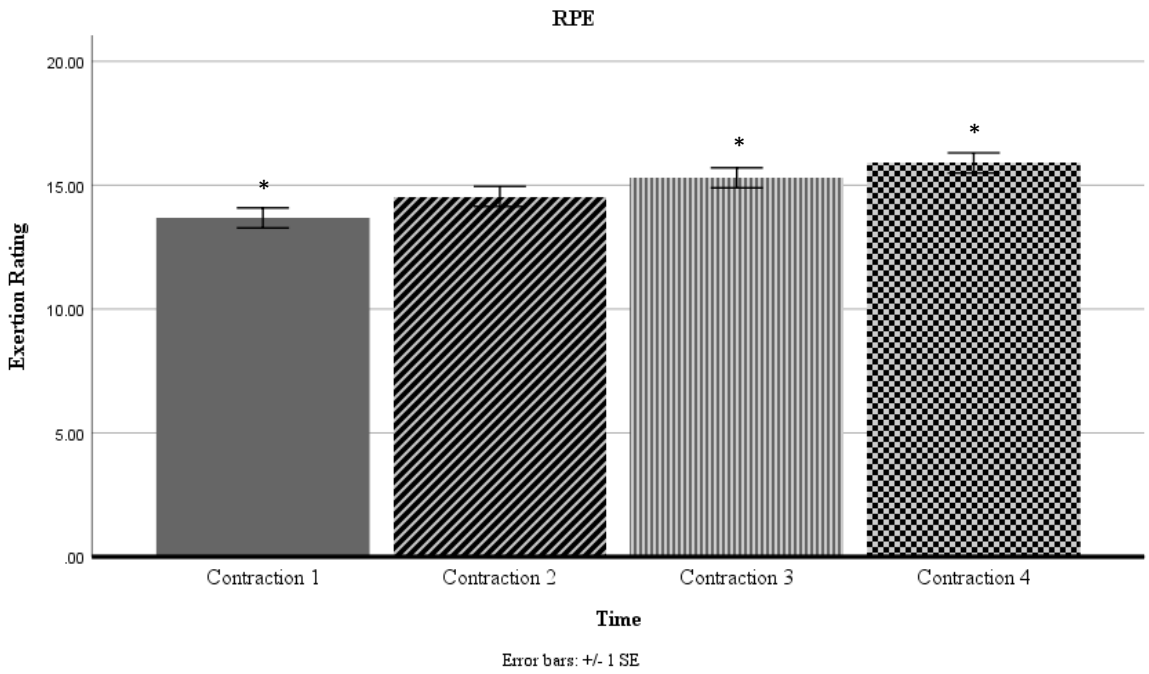


Figure 4: The acute effects of IHG on subjective ratings of RPE between contractions for both handgrip devices ($p<0.05$) Error bars ± SE. * significant differences for RPE between contractions ($p <0.05$).

2.5 Discussion

This study is the first to directly compare the IHG stimulus response when performed using a traditional computerized dynamometer and an inexpensive mechanical alternative. No study to date has examined the acute BP and HR response between traditional computerized handgrips and less expensive handgrip devices. Furthermore, the incorporation of surface EMG may allow for a more accurate understanding of muscular activation levels during the exercise which helps more comprehensively quantify the IHG stimulus. Lastly, incorporating subjective measures of pain and perceived exertion may provide evidence for the pragmatic implementation of cost-effective alternative devices for IHG exercise.

Acute Cardiovascular Effects During IHG Exercise

Currently, little is known about what happens to BP and HR during IHG exercise. Evidence has examined the acute cardiovascular effects regarding post-exercise hypotension (Ohler et al., 2013). Additionally, there is some work in hypertensive individuals examining peak cardiovascular changes (Olher et al., 2013). This study provides insight into the acute BP and HR response during the IHG exercise in a young, normotensive population using different IHG devices. As stated above, no statistically significant differences were observed in terms of SBP or HR between the two handgrip devices. In contrast, significant DBP differences were found, with the computerized device eliciting a greater response than the mechanical device.

Acute Effects on Muscular Activation During IHG Exercise

Through surface EMG, the present study examined the extent of muscular activation in the upper extremity during the IHG exercise. Muscular activation was similar for 3 of the 5 muscles tested during contractions using the mechanical handgrip device. The one muscle that was significant was the ECU. The reason for this muscle being activated more could be due to differences inherent to the mechanical handgrip device itself. The two devices differed in design which could have resulted in more activation of the ECU compared to the computerized device.

There is evidence to suggest that joint angle may influence EMG readings (Rabbi et al., 2017; Duque et al., 1995). As muscles change in length as a result to movement of joint angle, both force production and EMG values may be affected (Rabbi et al., 2017; Duque et al., 1995). Participants were instructed to keep their arm at a 90-degree angle, however, without the limb being mechanically fixed in that position, slight perturbations in the wrist or arm posture could have led to changes in EMG values. Moreover, the MVC trial was completed in the same fixed posture as the participants were in during testing. By doing this, the muscles may not have been

able to contract with their maximal effort. Therefore, MVC scores were not representative of the full activation of each muscle but perhaps just a peak EMG score during the maximal forces obtained whilst in that determined posture. This is apparent in the values being numerous times greater than 100% MVC. Despite this limitation, both trials on each device utilized the same fixed posture and 30% of the force during the maximum voluntary contraction was used to determine exercise intensity, and not 30% of the peak EMG activation. Therefore, the EMG values represent the percentage of peak muscle activation during the exercise relative to the original activation in the fixed 90-degree posture during the MVC trial and not the absolute peak EMG activation of the muscle itself.

Acute Effects on Perceived Subjective Pain and Exertion

To date, no IHG study has simultaneously investigated pain and ratings of perceived exertion during an exercise bout. Previous literature has shown that high intensity and/or painful past physical activity can act as a deterrent for future exercise (Lee et al., 2016).

The findings suggest that the mechanical handgrip device is no more painful and required a similar level of exertion compared to the computerized device. This may be the result of fatigue setting in as this trend still held true despite randomization of device order. Moreover, upon completing the final 30-minute rest period, all participants were asked to state which handgrip device they preferred. All 20 participants reported that they preferred the computerized handgrip device. This subjective finding suggests that the mechanical device may not be an ideal device to implement as an alternative IHG training device.

Limitations

Although the findings provide compelling evidence of the mechanical handgrip being a comparable stimulus to the computerized device, it is important to acknowledge several study limitations. The mechanical handgrip did not provide feedback to participants regarding their ability to maintain 30% MVC (e.g., squeezing too hard or not hard enough). Therefore, participants may have been exerting more (or less) than the target intensity. This may have resulted in participants being able to achieve the same stimulus at a lower exercise intensity.

Another limitation to this investigation was the inherit design differences of the two devices. The computerized device utilizes a force transducer within the handle, which provides little resistance to the participant. In contrast, the mechanical device utilizes a spring in order to generate resistance.

Significance and Future Directions

The findings of this study appear to be promising as they suggest that a cost-effective mechanical device may be a similar stimulus to the traditional computerized dynamometer. However, due to the low observed power from this study, the findings should be interpreted with caution and future studies should aim to replicate these acute findings in a large-scale, adequately powered investigation, and determine the effects of training.

2.6 References

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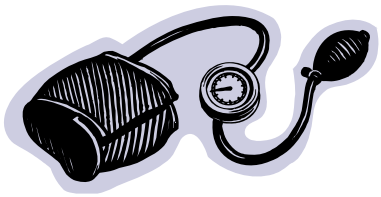
Appendix A: Recruitment Poster and Email Template:

Do you think you have normal blood pressure?



If so, you may be eligible to participate in a study examining the effects of a bout of handgrip exercise on blood pressure, heart rate and muscular activity of the arm using a computerized handgrip device vs a mechanical handgrip device. This study requires about 3 hours of your time split over 2 days (~0.5 hours for day 1 and ~2.5 hours for day 2)

If you are interested and would like more information, please call: (519) -253-3000 ext. 4979 or email: uwhandgrip@gmail.com

5	5	5	5	5	5	5	5	5	5	5	5	5
1	1	1	1	1	1	1	1	1	1	1	1	1
9	9	9	9	9	9	9	9	9	9	9	9	9
-	-	-	-	-	-	-	-	-	-	-	-	-
2	2	2	2	2	2	2	2	2	2	2	2	2
5	5	5	5	5	5	5	5	5	5	5	5	5
3	3	3	3	3	3	3	3	3	3	3	3	3
-	-	-	-	-	-	-	-	-	-	-	-	-
3	3	3	3	3	3	3	3	3	3	3	3	3
0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0
ext.	ext.	ext.	ext.	ext.	ext.	ext.	ext.	ext.	ext.	ext.	ext.	ext.
4979	4979	4979	4979	4979	4979	4979	4979	4979	4979	4979	4979	4979

This study has been cleared by the University of Windsor's Research Ethics Board

Email/Class recruitment script:

"Attention anyone between the age of 18 and 30 years. If you think you have normal blood pressure, you may be eligible to participate in a research study being conducted by researchers at the University of Windsor. We are investigating the effects of isometric handgrip exercise on your blood pressure, heart rate and arm muscles. For more information please contact Nic or at 519-253-3000 ex. 4979 or caruanan@uwindsor.ca”

Appendix B: Computerized Handgrip Device

Computerized Digital Dynamometer (IBX H-101, Zona Health, Boise, ID, USA)

Appendix C: Mechanical Handgrip Device

Mechanical Handgrip (Item code: 239247305, www.dhgate.com)

Appendix D: Informed Consent

CONSENT TO PARTICIPATE IN RESEARCH

Title of Study*: Comparative analysis of a bout of isometric hand grip using a traditional (computerized) or mechanical handgrip*

You are asked to participate in a research study conducted by Mr. Nic Caruana from the Faculty of Human Kinetics at the University of Windsor.

If you have any questions or concerns about the research, please feel to contact co-investigators Dr. Kevin Milne, PhD (kjmilne@uwindsor.ca), Dave Andrews, PhD (dandrews@uwindsor.ca) or Cheri McGowan, PhD (mcgowanc@uwindsor.ca).

PURPOSE OF THE STUDY

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Visit 2 (approximately 1 hour):
At least 24 hours after visit 1, you will meet with the study investigators for a familiarization session. You will be asked to have refrained from vigorous physical activity over the previous 24 hours, will be tested 2 hours post-prandial and at least 12 hours post-caffeine. To minimize the effects of a full bladder on BP, participants will be asked to void prior to familiarization session. If you are still interested in being a participant, you will be asked to provide on-going consent by initialling the visit 2 consent section of the consent form.

You will then be given 10 minutes of seated rest after which 4 resting blood pressure values will be taken. You will be seated for the duration of the familiarization period, with both your right and left forearms resting on a table in front of you at an approximate 90-degree angle. You will be outfitted with the necessary equipment to measure your BP, heart rate (HR) and electrical activity of the muscles through electromyography (EMG).

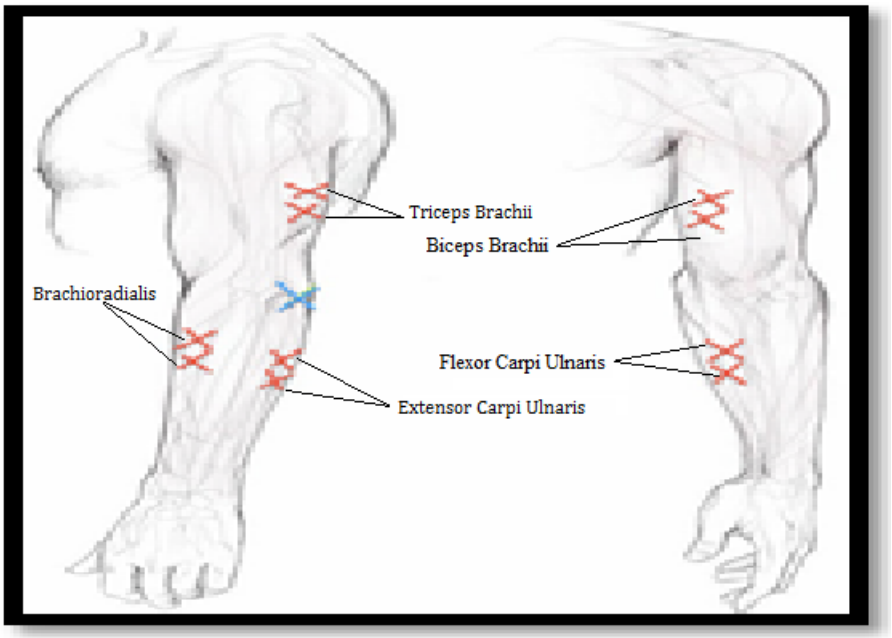
You will then be asked to perform 3 maximal 2 second squeezes separated by 1-minute rest between squeezes on either the computerized handgrip device or the mechanical handgrip device. Following the third squeeze you will complete one 2-minute sustained squeeze at an intensity of 30 % of your hardest squeeze during which blood pressure and heart rate measurements will be taken each minute. After you have completed the 2-minute squeeze, you will be asked to rate how much you feel you exerted yourself on a Borg 6-20 rating of perceived exertion scale, as well as asked to rank any discomfort that they may experience on a 1-10 numeric pain scale.

You will then be given a 10-minute rest period and then will be asked to complete the same protocol utilizing the other handgrip device. Once completed if the you are still interested in engaging in the study, visit 3 will be scheduled at least 24 hours after this session.

Visit 3 (approximately 2.5 hours):
If you are still interested in participating in the study, you will visit the lab at least 24 hours following Visit 1.

You will be asked not to exercise vigorously (e.g., exercise that causes you to breath really hard and sweat heavily) for 24 hours before the testing day, and to avoid caffeine for at least 12 hours before. All testing will take place in the morning at least 2 hours following your last meal, in a quiet, temperature-controlled room. You will be asked to go to the washroom before testing, as a full bladder can increase your BP.

You will be seated for the duration of the testing period, with both your right and left forearms resting on a table in front of you at an approximate 90-degree angle. You will be outfitted with the necessary equipment to measure your BP, heart rate (HR) and electrical activity of the muscles through electromyography (EMG). These measures will be collected continuously for 10 minutes prior to, during, and for at least 30 minutes following each IHG bout. Specifically, BP will be measured each minute using the device described above. Beat-to-beat HR will be acquired via standard 3-lead echocardiography (ECG) for later calculation of HR and offline assessment of its variability (heart rate variability, an indirect assessment of the nervous system). On your non-dominant arm, EMG data will be obtained by attaching two electrodes to the largest part of each muscle, parallel to the primary fascicle direction of the following arm muscles: forearm muscles - flexor carpi ulnaris, brachioradialis, extensor carpi ulnaris, brevis/longus; upper arm muscles - biceps brachii ("biceps") and triceps brachii ("triceps"). The skin to which the electrodes will be attached will be shaved with a disposable safety razor (if needed) and then cleaned with an alcohol swab prior to attaching the electrodes. A low allergy surgical tape will be placed over the electrodes and adhered to the skin to minimize movement during testing.



Next, you will be given 10 minutes of seated rest. Following the rest period, BP measurements will be taken in order to establish your baseline measurements. Following these measurements, your maximum voluntary contraction (MVC; hardest squeeze) will be calculated using each handgrip (traditional, computerized IHG and store-bought mechanical IHG) in order to determine the intensity to which the IHG bouts will be performed.

Next, you will be asked perform, in random order, an IHG protocol on the traditional, computerized IHG dynamometer and the store-bought mechanical grip. The protocols will be separated by a 30-minute rest period (minimum) or until your BP has returned to near pre-exercise values. During the 30-minute rest period you will have your blood pressure taken every 5 minutes

to make sure your BP is returning to pre-exercising values. In both conditions, the IHG bout will consist of four, 2-minute unilateral (non-dominant arm) contractions at 30% of the MVC, each separated by 4-minute rest period. Following each 2-minute contraction, you will be asked to rate your level of perceived exertion using a 6-20 scale as well as rate any discomfort that you may be feeling on a 1-10 scale. Upon completion of the final bout of handgrip, there will a final stabilization period to ensure that your BP returns to near pre-exercise values. Finally, upon completion of the final 30- minute rest period, you will be asked to state which handgrip device you preferred.

POTENTIAL RISKS AND DISCOMFORTS

You may experience numbness and/or tingling in the limb while the BP cuff inflates but this should disappear when the cuff is released. The sticker-electrodes used to measure your HR and muscle activity may cause a skin irritation however, this risk is minimal. Tendonitis is possible from the IHG bouts, but as this occurs during a single session (and not for a training intervention) and proper technique will be enforced by study investigators during the session, this risk is minimal.

Please contact one of the study investigators if you feel any adverse effects from completing any portion of the study, and/or if you have any questions or concerns. If you experience any adverse effects during any testing procedure, first line response will be provided.

POTENTIAL BENEFITS TO PARTICIPANTS AND/OR TO SOCIETY

You may not experience any physical direct benefit by participating in this single session study. However, if we prove our theories, evidence of the equivalence of a mechanical handgrip in comparison to the traditional, more expensive computerized IHG may lay the groundwork for future studies investigating the use of affordable, commercially available handgrips as a means to lower BP

COMPENSATION FOR PARTICIPATION

All participants will receive a Kinesiology Research T-shirt upon their completion of the study**.**

CONFIDENTIALITY

Any information that is obtained in connection with this study that can identify you will remain confidential.

To ensure your confidentiality, following your consent, you will be assigned an identification number. Your name will not be mentioned in any publication or presentation, and you will be identified with only your identification number on all collection tools (electronic or otherwise). All paper data will be stored in the locked laboratory (PACR Lab, Room #240, Human Kinetics Building, University of Windsor). Information stored on computer will be password-accessible only. With respect to final disposal, all paper records (including medical and physical activity readiness questionnaires) will be shredded after 5 years.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not, and your participation or lack of it will not influence your participation in another study. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may also refuse to answer any questions you do not wish to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. In any of the cases described above, you will still receive a Kinesiology Research T-Shirt.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE PARTICIPANTS

Results of the study will be posted on the University of Windsor's Research Ethics Board (REB) website (http://www.uwindsor.ca/reb) at the completion of the study.

SUBSEQUENT USE OF DATA

These data may be used in subsequent studies, in publications and in presentations however your privacy will be upheld with the use of your unique subject identification number under all circumstances. Data may be used as a foundation for future study of store-bought, readily available IHG, and/or merged with other data sets for comparison.

RIGHTS OF RESEARCH PARTICIPANTS

If you have questions regarding your rights as a research participant, contact: Research Ethics Coordinator, University of Windsor, Windsor, Ontario, N9B 3P4; Telephone: 519-253-3000, ext. 3948; e-mail: ethics@uwindsor.ca

SIGNATURE OF RESEARCH PARTICIPANT/LEGAL REPRESENTATIVE

I understand the information provided for the study: *Comparative analysis of an acute bout of isometric hand grip using a traditional (computerized) or mechanical handgrip* as described herein. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

_____

Name of Participant

_____ _____

Signature of Participant Date

Ongoing consent for Visit 2:

_____ _____

Initial of Participant Date

Ongoing consent for Visit 3:

_____ _____

Initial of Participant Date

SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

_____ _____

Signature of Investigator Date

Appendix E: Letter of Information

LETTER OF INFORMATION FOR CONSENT TO PARTICIPATE IN RESEARCH

Title of Study*: Comparative analysis of a bout of isometric hand grip using a traditional (computerized) or mechanical handgrip*

You are asked to participate in a research study conducted by Mr. Nic Caruana from the Faculty of Human Kinetics at the University of Windsor.

If you have any questions or concerns about the research, please feel to contact co-investigators Dr. Kevin Milne, PhD (kjmilne@uwindsor.ca), Dave Andrews, PhD (dandrews@uwindsor.ca) or Cheri McGowan, PhD (mcgowanc@uwindsor.ca).

PURPOSE OF THE STUDY

Our research group has shown that isometric (constant squeeze) exercise training using a computerized handgrip (isometric handgrip, IHG) lowers resting blood pressure (BP) in younger and older people, and in those with and without high BP. The computerized IHG is very expensive, and so we would like to see if an inexpensive mechanical handgrip that you can buy at the store may work just as well. Before we can do that, we need to figure out if the mechanical IHG causes your body to respond the same way as the computerized one.

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At this time, one of the study investigators will explain all parts of the study. If you are still interested in participating in the study, you will sign the consent form and fill out brief medical questionnaires. If you are still eligible to participate, you will then have your BP measured in your upper arm, similar to how it is taken at a doctor's office in brief, your resting BP will be measured in your upper dominant arm after 10 minutes of seated rest. Your BP will be measured 4 times, with 2 minutes of rest between measures. If you are eligible to participate, you will then have an opportunity to practice all parts of the study.

Visit 2 (approximately 1 hour):

At least 24 hours after visit 1, you will meet with the study investigators for a familiarization session. You will be asked to have refrained from vigorous physical activity over the previous 24 hours, will be tested 2 hours post-prandial and at least 12 hours post-caffeine. To minimize the effects of a full bladder on BP, participants will be asked to void prior to familiarization session. If you are still interested in being a participant, you will be asked to provide on-going consent by initialling the visit 2 consent section of the consent form.

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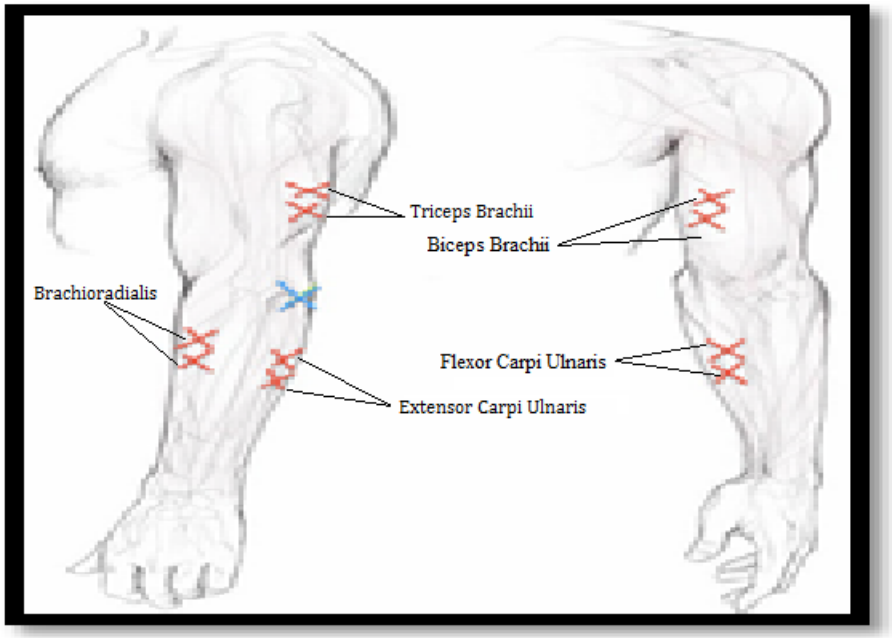

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SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

_____ _____

Signature of Investigator Date

Appendix F: Intake Medical Questionnaire and Physical Activity Readiness Questionnaire (PAR-Q)

Participant code: _____

Height: _____ Mass: _____ Date of Birth (Month/Yr) _____

Phone (_____)_____ Postal Code _____

FOR EMERGENCY NOTIFY: Name_____

Relationship _____

Address _____ Phone _____

Family Doctor's Name _____

Date of Last Physical _____

Please Check: Yes or No

	Yes	No
1. Have you ever been hospitalized?	☐	☐

If yes, please specify?

_____

_____

Have you ever had surgery? ☐ ☐

If yes, please specify?

_____

_____

2. Are you presently taking any medications or pills (including aspirin and other over the-counter medication)?

☐ ☐

If yes, please specify?

_____

_____

Are you presently taking any vitamins, supplements, and/or herbal supplements?

☐ ☐

3. Do you have any allergies (medicine, food, bees or other stinging insects)? ☐ ☐

If yes, please specify?

_____

_____

4. Have you ever passed out during or after exercise? ☐ ☐

Have you ever been dizzy during or after exercise? ☐ ☐

Have you ever had chest pain during or after exercise? ☐ ☐

Yes No

Do you have high blood pressure (hypertension) or low blood pressure (hypotension)? ☐ ☐

Have you ever been told that you have a kidney problem? ☐ ☐

Have you ever been told that you have joint instability? ☐ ☐

Have you ever been told that you have a stomach problem? ☐ ☐

Have you ever been told that you have a heart problem? ☐ ☐

Have you ever been told that you have a heart murmur? ☐ ☐

Do you have a machine that regulated your heart beat? ☐ ☐

Have you ever had racing of your heart or skipped heartbeats? ☐ ☐

Has anyone in your family died of heart problems or a sudden death before age 50? ☐ ☐

5. Do you have any skin problems (itching, rashes, acne)? ☐ ☐

If you get a cut, does it take you a long time to stop bleeding? ☐ ☐

If you experience a blow to a muscle, do you bruise easily? ☐ ☐

6. Do you have Diabetes? ☐ ☐

7. Do you have Asthma or any other breathing problems? ☐ ☐
   If yes, please specify?

_____

_____

8. Do you have any type of cardiovascular disease? ☐ ☐

If yes, please specify?

_____

_____

9. Have you had any other medical problems (infectious mononucleosis, etc.)? ☐ ☐

10. Have you had any medical problems since your last physical? ☐ ☐

11. Do you smoke? ☐ ☐

12. Do you aerobically exercise (e.g., walking) for
≥ 30 minutes, > 2 times per week? ☐ ☐

13. Do you currently take any birth control medications? ☐ ☐

If yes, please specify?

_____

_____

14. Date of last menstrual cycle_____

Please explain any physical limitations that may prevent you from completing this study:

Physical Activity Readiness Questionnaire - PAR-Q (revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO		
☐	☐	1.	**Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**
☐	☐	2.	**Do you feel pain in your chest when you do physical activity?**
☐	☐	3.	**In the past month, have you had chest pain when you were not doing physical activity?**
☐	☐	4.	**Do you lose your balance because of dizziness or do you ever lose consciousness?**
☐	☐	5.	**Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	6.	**Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	7.	**Do you know of any other reason why you should not do physical activity?**

If you answered YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

→ **DELAY BECOMING MUCH MORE ACTIVE:**

- if you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- if you are or may be pregnant – talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____ DATE _____

SIGNATURE OF PARENT or GUARDIAN (for participants under the age of majority) _____ WITNESS _____

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

Supported by: Health Canada Santé Canada

continued on other side...

Appendix G: Blood Pressure Oscillometric Device

(Dinamap Carescape v100, Critikon, Tampa, Florida, USA)

Appendix H: EMG Equipment and Electrodes

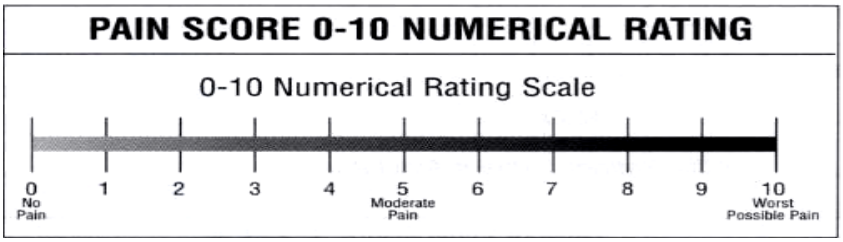
(PUN-96, Bortec Biological Limited, Calgary, Alberta, Canada)

(Covidien llc. Mansfield, MA, USA)

Appendix I: Borg Rating of Perceived Exertion Scale (RPE)

Rating	Perceived Exertion
6	No exertion
7	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Extremely hard
20	Maximal exertion

Appendix J: Pain Assessment: Numerical Rating Scale for Pain (NRS Pain)



Appendix K: Statistical Data for Chapter 2

1. SBP (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Device	Sphericity Assumed	762.066	1	762.066	2.798	.113
	Greenhouse-Geisser	762.066	1.000	762.066	2.798	.113
	Huynh-Feldt	762.066	1.000	762.066	2.798	.113
	Lower-bound	762.066	1.000	762.066	2.798	.113
Device * SEX	Sphericity Assumed	1255.811	1	1255.811	4.610	.047
	Greenhouse-Geisser	1255.811	1.000	1255.811	4.610	.047
	Huynh-Feldt	1255.811	1.000	1255.811	4.610	.047
	Lower-bound	1255.811	1.000	1255.811	4.610	.047
Device * Device_Starting_Order	Sphericity Assumed	23.131	1	23.131	.085	.774
	Greenhouse-Geisser	23.131	1.000	23.131	.085	.774

	Huynh-Feldt	23.131	1.000	23.131	.085	.774
	Lower-bound	23.131	1.000	23.131	.085	.774
Error(Device)	Sphericity Assumed	4630.891	17	272.405		
	Greenhouse-Geisser	4630.891	17.000	272.405		
	Huynh-Feldt	4630.891	17.000	272.405		
	Lower-bound	4630.891	17.000	272.405		
Contraction	Sphericity Assumed	63.998	3	21.333	.230	.875
	Greenhouse-Geisser	63.998	1.888	33.902	.230	.783
	Huynh-Feldt	63.998	2.366	27.049	.230	.830
	Lower-bound	63.998	1.000	63.998	.230	.637
Contraction * SEX	Sphericity Assumed	412.972	3	137.657	1.486	.229
	Greenhouse-Geisser	412.972	1.888	218.764	1.486	.241
	Huynh-Feldt	412.972	2.366	174.547	1.486	.237
	Lower-bound	412.972	1.000	412.972	1.486	.239

Contraction * Device_Starting_Order	Sphericity Assumed	302.939	3	100.980	1.090	.362
	Greenhouse-Geisser	302.939	1.888	160.476	1.090	.345
	Huynh-Feldt	302.939	2.366	128.040	1.090	.354
	Lower-bound	302.939	1.000	302.939	1.090	.311
Error(Contraction)	Sphericity Assumed	4723.402	51	92.616		
	Greenhouse-Geisser	4723.402	32.092	147.184		
	Huynh-Feldt	4723.402	40.221	117.435		
	Lower-bound	4723.402	17.000	277.847		
Device * Contraction	Sphericity Assumed	355.040	3	118.347	1.308	.282
	Greenhouse-Geisser	355.040	1.785	198.856	1.308	.283
	Huynh-Feldt	355.040	2.216	160.252	1.308	.284
	Lower-bound	355.040	1.000	355.040	1.308	.269
Device * Contraction * SEX	Sphericity Assumed	301.969	3	100.656	1.113	.353
	Greenhouse-Geisser	301.969	1.785	169.132	1.113	.336

	Huynh-Feldt	301.969	2.216	136.298	1.113	.344
	Lower-bound	301.969	1.000	301.969	1.113	.306
Device * Contraction * Device_Starting_Order	Sphericity Assumed	242.194	3	80.731	.892	.451
	Greenhouse-Geisser	242.194	1.785	135.652	.892	.410
	Huynh-Feldt	242.194	2.216	109.317	.892	.427
	Lower-bound	242.194	1.000	242.194	.892	.358
Error(Device*Contraction)	Sphericity Assumed	4613.551	51	90.462		
	Greenhouse-Geisser	4613.551	30.352	152.002		
	Huynh-Feldt	4613.551	37.664	122.493		
	Lower-bound	4613.551	17.000	271.385		

Sex* Device interaction

ANOVA

SBP

	Sum of Squares	df	Mean Square	F
Between Groups	2105.519	3	701.840	5.061

Within Groups	21635.225	156	138.687	
Total	23740.744	159		

ANOVA

SBP

	Sig.
Between Groups	.002
Within Groups	
Total	

Post Hoc Tests

Multiple Comparisons

Dependent Variable: SBP

Tukey HSD

(I) Gender2	(J) Gender2	Mean Difference (I-J)	Std. Error	Sig.
Male Mech	Male Comp	9.18182*	2.51077	.002
	Female Mech	7.71717*	2.64658	.021
	Female Comp	5.30051	2.64658	.191
Male Comp	Male Mech	-9.18182*	2.51077	.002
	Female Mech	-1.46465	2.64658	.946
	Female Comp	-3.88131	2.64658	.460

Female Mech	Male Mech	-7.71717*	2.64658	.021
	Male Comp	1.46465	2.64658	.946
	Female Comp	-2.41667	2.77576	.820
Female Comp	Male Mech	-5.30051	2.64658	.191
	Male Comp	3.88131	2.64658	.460
	Female Mech	2.41667	2.77576	.820

Multiple Comparisons

Dependent Variable: SBP

Tukey HSD

		95% Confidence Interval	
(I) Gender2	(J) Gender2	Lower Bound	Upper Bound
Male Mech	Male Comp	2.6615	15.7021
	Female Mech	.8442	14.5902
	Female Comp	-1.5725	12.1735
Male Comp	Male Mech	-15.7021	-2.6615
	Female Mech	-8.3377	5.4084
	Female Comp	-10.7543	2.9917
Female Mech	Male Mech	-14.5902	-.8442
	Male Comp	-5.4084	8.3377
	Female Comp	-9.6251	4.7918
Female Comp	Male Mech	-12.1735	1.5725
	Male Comp	-2.9917	10.7543
	Female Mech	-4.7918	9.6251

*. The mean difference is significant at the 0.05 level.

2. DBP (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Device	Sphericity Assumed	1522.861	1	1522.861	5.902	.027
	Greenhouse-Geisser	1522.861	1.000	1522.861	5.902	.027
	Huynh-Feldt	1522.861	1.000	1522.861	5.902	.027
	Lower-bound	1522.861	1.000	1522.861	5.902	.027
Device * SEX	Sphericity Assumed	1748.801	1	1748.801	6.777	.019
	Greenhouse-Geisser	1748.801	1.000	1748.801	6.777	.019
	Huynh-Feldt	1748.801	1.000	1748.801	6.777	.019
	Lower-bound	1748.801	1.000	1748.801	6.777	.019

Device * Device_Starting_Order	Sphericity Assumed	670.925	1	670.925	2.600	.125
	Greenhouse-Geisser	670.925	1.000	670.925	2.600	.125
	Huynh-Feldt	670.925	1.000	670.925	2.600	.125
	Lower-bound	670.925	1.000	670.925	2.600	.125
Error(Device)	Sphericity Assumed	4386.598	17	258.035		
	Greenhouse-Geisser	4386.598	17.000	258.035		
	Huynh-Feldt	4386.598	17.000	258.035		
	Lower-bound	4386.598	17.000	258.035		
Contraction	Sphericity Assumed	261.820	3	87.273	1.095	.360
	Greenhouse-Geisser	261.820	2.026	129.204	1.095	.347

		Huynh-Feldt	261.820	2.573	101.754	1.095	.355
		Lower-bound	261.820	1.000	261.820	1.095	.310
	Contraction * SEX	Sphericity Assumed	458.180	3	152.727	1.916	.139
		Greenhouse-Geisser	458.180	2.026	226.105	1.916	.162
		Huynh-Feldt	458.180	2.573	178.068	1.916	.149
		Lower-bound	458.180	1.000	458.180	1.916	.184
	Contraction * Device_Starting_Order	Sphericity Assumed	138.511	3	46.170	.579	.631
		Greenhouse-Geisser	138.511	2.026	68.353	.579	.568
		Huynh-Feldt	138.511	2.573	53.831	.579	.606
		Lower-bound	138.511	1.000	138.511	.579	.457

	Error(Contraction)	Sphericity Assumed	4064.547	51	79.697		
		Greenhouse-Geisser	4064.547	34.449	117.988		
		Huynh-Feldt	4064.547	43.742	92.921		
		Lower-bound	4064.547	17.000	239.091		
	Device * Contraction	Sphericity Assumed	476.145	3	158.715	1.699	.179
		Greenhouse-Geisser	476.145	1.871	254.542	1.699	.200
		Huynh-Feldt	476.145	2.341	203.428	1.699	.192
		Lower-bound	476.145	1.000	476.145	1.699	.210
	Device * Contraction * SEX	Sphericity Assumed	171.293	3	57.098	.611	.611
		Greenhouse-Geisser	171.293	1.871	91.571	.611	.538

		Huynh-Feldt	171.293	2.341	73.183	.611	.572
		Lower-bound	171.293	1.000	171.293	.611	.445
	Device * Contraction * Device_Starting_Order	Sphericity Assumed	716.250	3	238.750	2.556	.065
		Greenhouse-Geisser	716.250	1.871	382.899	2.556	.097
		Huynh-Feldt	716.250	2.341	306.011	2.556	.082
		Lower-bound	716.250	1.000	716.250	2.556	.128
	Error(Device*Contraction)	Sphericity Assumed	4763.308	51	93.398		
		Greenhouse-Geisser	4763.308	31.800	149.789		
		Huynh-Feldt	4763.308	39.790	119.710		
		Lower-bound	4763.308	17.000	280.195		

Device * Sex interaction

ANOVA

DBP

	Sum of Squares	df	Mean Square	F
Between Groups	1506.266	3	502.089	3.331
Within Groups	23516.977	156	150.750	
Total	25023.244	159		

ANOVA

DBP

	Sig.
Between Groups	.021
Within Groups	
Total	

Post Hoc Tests

Multiple Comparisons

Dependent Variable: DBP

Tukey HSD

(I) Gender2	(J) Gender2	Mean Difference (I-J)	Std. Error	Sig.
Male Mech	Male Comp	4.43182	2.61768	.331
	Female Mech	8.28030*	2.75928	.016
	Female Comp	1.78030	2.75928	.917
Male Comp	Male Mech	-4.43182	2.61768	.331
	Female Mech	3.84848	2.75928	.505
	Female Comp	-2.65152	2.75928	.772
Female Mech	Male Mech	-8.28030*	2.75928	.016
	Male Comp	-3.84848	2.75928	.505
	Female Comp	-6.50000	2.89396	.116
Female Comp	Male Mech	-1.78030	2.75928	.917
	Male Comp	2.65152	2.75928	.772
	Female Mech	6.50000	2.89396	.116

Multiple Comparisons

Dependent Variable: DBP

Tukey HSD

		95% Confidence Interval	
(I) Gender2	(J) Gender2	Lower Bound	Upper Bound
Male Mech	Male Comp	-2.3661	11.2298
	Female Mech	1.1146	15.4460
	Female Comp	-5.3854	8.9460
Male Comp	Male Mech	-11.2298	2.3661
	Female Mech	-3.3172	11.0142
	Female Comp	-9.8172	4.5142

Female Mech	Male Mech	-15.4460	-1.1146
	Male Comp	-11.0142	3.3172
	Female Comp	-14.0154	1.0154
Female Comp	Male Mech	-8.9460	5.3854
	Male Comp	-4.5142	9.8172
	Female Mech	-1.0154	14.0154

*. The mean difference is significant at the 0.05 level.

3. HR (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Device	Sphericity Assumed	207.047	1	207.047	1.958	.180
	Greenhouse-Geisser	207.047	1.000	207.047	1.958	.180
	Huynh-Feldt	207.047	1.000	207.047	1.958	.180
	Lower-bound	207.047	1.000	207.047	1.958	.180
Device * SEX	Sphericity Assumed	72.928	1	72.928	.690	.418
	Greenhouse-Geisser	72.928	1.000	72.928	.690	.418
	Huynh-Feldt	72.928	1.000	72.928	.690	.418
	Lower-bound	72.928	1.000	72.928	.690	.418
Device * Device_Starting_Order	Sphericity Assumed	248.685	1	248.685	2.352	.144
	Greenhouse-Geisser	248.685	1.000	248.685	2.352	.144
	Huynh-Feldt	248.685	1.000	248.685	2.352	.144
	Lower-bound	248.685	1.000	248.685	2.352	.144
Error(Device)	Sphericity Assumed	1797.714	17	105.748		
	Greenhouse-Geisser	1797.714	17.000	105.748		
	Huynh-Feldt	1797.714	17.000	105.748		
	Lower-bound	1797.714	17.000	105.748		

Contraction	Sphericity Assumed	42.861	3	14.287	.681	.568
	Greenhouse-Geisser	42.861	2.469	17.360	.681	.541
	Huynh-Feldt	42.861	3.000	14.287	.681	.568
	Lower-bound	42.861	1.000	42.861	.681	.421
Contraction * SEX	Sphericity Assumed	35.288	3	11.763	.561	.643
	Greenhouse-Geisser	35.288	2.469	14.293	.561	.611
	Huynh-Feldt	35.288	3.000	11.763	.561	.643
	Lower-bound	35.288	1.000	35.288	.561	.464
Contraction * Device_Starting_Order	Sphericity Assumed	34.793	3	11.598	.553	.649
	Greenhouse-Geisser	34.793	2.469	14.093	.553	.616
	Huynh-Feldt	34.793	3.000	11.598	.553	.649
	Lower-bound	34.793	1.000	34.793	.553	.467
Error(Contraction)	Sphericity Assumed	1070.192	51	20.984		
	Greenhouse-Geisser	1070.192	41.971	25.499		
	Huynh-Feldt	1070.192	51.000	20.984		
	Lower-bound	1070.192	17.000	62.952		
Device * Contraction	Sphericity Assumed	86.789	3	28.930	1.383	.258
	Greenhouse-Geisser	86.789	2.542	34.146	1.383	.262
	Huynh-Feldt	86.789	3.000	28.930	1.383	.258
	Lower-bound	86.789	1.000	86.789	1.383	.256
Device * Contraction * SEX	Sphericity Assumed	56.221	3	18.740	.896	.450
	Greenhouse-Geisser	56.221	2.542	22.120	.896	.437
	Huynh-Feldt	56.221	3.000	18.740	.896	.450
	Lower-bound	56.221	1.000	56.221	.896	.357
Device * Contraction * Device_Starting_Order	Sphericity Assumed	87.080	3	29.027	1.388	.257

	Greenhouse-Geisser	87.080	2.542	34.261	1.388	.261
	Huynh-Feldt	87.080	3.000	29.027	1.388	.257
	Lower-bound	87.080	1.000	87.080	1.388	.255
Error(Device*Contraction)	Sphericity Assumed	1066.612	51	20.914		
	Greenhouse-Geisser	1066.612	43.209	24.685		
	Huynh-Feldt	1066.612	51.000	20.914		
	Lower-bound	1066.612	17.000	62.742		

4. BR EMG (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
HandgripDevice	Sphericity Assumed	1785.525	1	1785.525	3.845	.066
	Greenhouse-Geisser	1785.525	1.000	1785.525	3.845	.066
	Huynh-Feldt	1785.525	1.000	1785.525	3.845	.066
	Lower-bound	1785.525	1.000	1785.525	3.845	.066
HandgripDevice * Sex	Sphericity Assumed	423.278	1	423.278	.912	.353
	Greenhouse-Geisser	423.278	1.000	423.278	.912	.353
	Huynh-Feldt	423.278	1.000	423.278	.912	.353
	Lower-bound	423.278	1.000	423.278	.912	.353
HandgripDevice * Order_Starting_Device	Sphericity Assumed	243.147	1	243.147	.524	.479
	Greenhouse-Geisser	243.147	1.000	243.147	.524	.479

	Huynh-Feldt	243.147	1.000	243.147	.524	.479
	Lower-bound	243.147	1.000	243.147	.524	.479
Error(HandgripDevice)	Sphericity Assumed	7893.847	17	464.344		
	Greenhouse-Geisser	7893.847	17.000	464.344		
	Huynh-Feldt	7893.847	17.000	464.344		
	Lower-bound	7893.847	17.000	464.344		
Contraction	Sphericity Assumed	164315.268	3	54771.756	2.474	.072
	Greenhouse-Geisser	164315.268	1.810	90764.900	2.474	.105
	Huynh-Feldt	164315.268	2.252	72964.854	2.474	.092
	Lower-bound	164315.268	1.000	164315.268	2.474	.134
Contraction * Sex	Sphericity Assumed	93406.283	3	31135.428	1.406	.252
	Greenhouse-Geisser	93406.283	1.810	51596.008	1.406	.259
	Huynh-Feldt	93406.283	2.252	41477.434	1.406	.258
	Lower-bound	93406.283	1.000	93406.283	1.406	.252
Contraction * Order_Starting_Device	Sphericity Assumed	130363.904	3	43454.635	1.962	.131
	Greenhouse-Geisser	130363.904	1.810	72010.756	1.962	.161
	Huynh-Feldt	130363.904	2.252	57888.614	1.962	.150
	Lower-bound	130363.904	1.000	130363.904	1.962	.179
Error(Contraction)	Sphericity Assumed	1129269.365	51	22142.537		

	Greenhouse-Geisser	1129269.365	30.776	36693.458		
	Huynh-Feldt	1129269.365	38.284	29497.446		
	Lower-bound	1129269.365	17.000	66427.610		
HandgripDevice * Contraction	Sphericity Assumed	8998.987	3	2999.662	3.721	.017
	Greenhouse-Geisser	8998.987	2.457	3663.004	3.721	.025
	Huynh-Feldt	8998.987	3.000	2999.662	3.721	.017
	Lower-bound	8998.987	1.000	8998.987	3.721	.071
HandgripDevice * Contraction * Sex	Sphericity Assumed	5250.664	3	1750.221	2.171	.103
	Greenhouse-Geisser	5250.664	2.457	2137.263	2.171	.117
	Huynh-Feldt	5250.664	3.000	1750.221	2.171	.103
	Lower-bound	5250.664	1.000	5250.664	2.171	.159
HandgripDevice * Contraction * Order_Starting_Device	Sphericity Assumed	3628.272	3	1209.424	1.500	.226
	Greenhouse-Geisser	3628.272	2.457	1476.874	1.500	.233
	Huynh-Feldt	3628.272	3.000	1209.424	1.500	.226
	Lower-bound	3628.272	1.000	3628.272	1.500	.237
Error(HandgripDevice*Contraction)	Sphericity Assumed	41117.455	51	806.225		
	Greenhouse-Geisser	41117.455	41.764	984.512		
	Huynh-Feldt	41117.455	51.000	806.225		
	Lower-bound	41117.455	17.000	2418.674		

5. BB EMG (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
HandgripDevice	Sphericity Assumed	5899.436	1	5899.436	6.410	.021
	Greenhouse-Geisser	5899.436	1.000	5899.436	6.410	.021
	Huynh-Feldt	5899.436	1.000	5899.436	6.410	.021
	Lower-bound	5899.436	1.000	5899.436	6.410	.021
HandgripDevice * Sex	Sphericity Assumed	3255.171	1	3255.171	3.537	.077
	Greenhouse-Geisser	3255.171	1.000	3255.171	3.537	.077
	Huynh-Feldt	3255.171	1.000	3255.171	3.537	.077
	Lower-bound	3255.171	1.000	3255.171	3.537	.077
HandgripDevice * Order_Starting_Device	Sphericity Assumed	2600.764	1	2600.764	2.826	.111
	Greenhouse-Geisser	2600.764	1.000	2600.764	2.826	.111
	Huynh-Feldt	2600.764	1.000	2600.764	2.826	.111
	Lower-bound	2600.764	1.000	2600.764	2.826	.111
Error(HandgripDevice)	Sphericity Assumed	15645.643	17	920.332		
	Greenhouse-Geisser	15645.643	17.000	920.332		
	Huynh-Feldt	15645.643	17.000	920.332		
	Lower-bound	15645.643	17.000	920.332		
Contraction	Sphericity Assumed	801034.265	3	267011.422	4.393	.008
	Greenhouse-Geisser	801034.265	1.007	795632.150	4.393	.051
	Huynh-Feldt	801034.265	1.134	706399.138	4.393	.045

	Lower-bound	801034.265	1.000	801034.265	4.393	.051
Contraction * Sex	Sphericity Assumed	459909.937	3	153303.312	2.522	.068
	Greenhouse-Geisser	459909.937	1.007	456808.339	2.522	.130
	Huynh-Feldt	459909.937	1.134	405575.638	2.522	.126
	Lower-bound	459909.937	1.000	459909.937	2.522	.131
Contraction * Order_Starting_Device	Sphericity Assumed	477939.820	3	159313.273	2.621	.061
	Greenhouse-Geisser	477939.820	1.007	474716.630	2.621	.124
	Huynh-Feldt	477939.820	1.134	421475.449	2.621	.119
	Lower-bound	477939.820	1.000	477939.820	2.621	.124
Error(Contraction)	Sphericity Assumed	3099652.686	51	60777.504		
	Greenhouse-Geisser	3099652.686	17.115	181102.874		
	Huynh-Feldt	3099652.686	19.277	160791.534		
	Lower-bound	3099652.686	17.000	182332.511		
HandgripDevice * Contraction	Sphericity Assumed	25709.080	3	8569.693	5.002	.004
	Greenhouse-Geisser	25709.080	1.076	23897.253	5.002	.036
	Huynh-Feldt	25709.080	1.226	20977.085	5.002	.030
	Lower-bound	25709.080	1.000	25709.080	5.002	.039
HandgripDevice * Contraction * Sex	Sphericity Assumed	15212.922	3	5070.974	2.960	.041
	Greenhouse-Geisser	15212.922	1.076	14140.803	2.960	.100
	Huynh-Feldt	15212.922	1.226	12412.842	2.960	.094
	Lower-bound	15212.922	1.000	15212.922	2.960	.103
HandgripDevice * Contraction * Order_Starting_Device	Sphericity Assumed	11990.838	3	3996.946	2.333	.085
	Greenhouse-Geisser	11990.838	1.076	11145.793	2.333	.143
	Huynh-Feldt	11990.838	1.226	9783.813	2.333	.138

	Lower-bound	11990.838	1.000	11990.838	2.333	.145
Error(HandgripDevice*Contraction)	Sphericity Assumed	87369.323	51	1713.124		
	Greenhouse-Geisser	87369.323	18.289	4777.179		
	Huynh-Feldt	87369.323	20.835	4193.423		
	Lower-bound	87369.323	17.000	5139.372		

6. ECU EMG (All Effects)
Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
HandgripDevice	Sphericity Assumed	1118.376	1	1118.376	9.534	.007
	Greenhouse-Geisser	1118.376	1.000	1118.376	9.534	.007
	Huynh-Feldt	1118.376	1.000	1118.376	9.534	.007
	Lower-bound	1118.376	1.000	1118.376	9.534	.007
HandgripDevice * Sex	Sphericity Assumed	322.342	1	322.342	2.748	.116

		Greenhouse-Geisser	322.342	1.000	322.342	2.748	.116
		Huynh-Feldt	322.342	1.000	322.342	2.748	.116
		Lower-bound	322.342	1.000	322.342	2.748	.116
	HandgripDevice * Order_Starting_Device	Sphericity Assumed	380.523	1	380.523	3.244	.089
		Greenhouse-Geisser	380.523	1.000	380.523	3.244	.089
		Huynh-Feldt	380.523	1.000	380.523	3.244	.089
		Lower-bound	380.523	1.000	380.523	3.244	.089
	Error(HandgripDevice)	Sphericity Assumed	1994.189	17	117.305		
		Greenhouse-Geisser	1994.189	17.000	117.305		
		Huynh-Feldt	1994.189	17.000	117.305		

	Lower-bound	1994.189	17.000	117.305		
Contraction	Sphericity Assumed	16105.306	3	5368.435	1.293	.287
	Greenhouse-Geisser	16105.306	1.172	13740.257	1.293	.277
	Huynh-Feldt	16105.306	1.355	11888.195	1.293	.281
	Lower-bound	16105.306	1.000	16105.306	1.293	.271
Contraction * Sex	Sphericity Assumed	21521.973	3	7173.991	1.728	.173
	Greenhouse-Geisser	21521.973	1.172	18361.492	1.728	.205
	Huynh-Feldt	21521.973	1.355	15886.530	1.728	.204
	Lower-bound	21521.973	1.000	21521.973	1.728	.206
Contraction * Order_Starting_Device	Sphericity Assumed	2697.329	3	899.110	.217	.884

		Greenhouse-Geisser	2697.329	1.172	2301.228	.217	.685
		Huynh-Feldt	2697.329	1.355	1991.044	.217	.719
		Lower-bound	2697.329	1.000	2697.329	.217	.648
	Error(Contraction)	Sphericity Assumed	211679.029	51	4150.569		
		Greenhouse-Geisser	211679.029	19.926	10623.186		
		Huynh-Feldt	211679.029	23.030	9191.277		
		Lower-bound	211679.029	17.000	12451.708		
	HandgripDevice * Contraction	Sphericity Assumed	4026.511	3	1342.170	3.109	.034
		Greenhouse-Geisser	4026.511	1.233	3266.249	3.109	.085
		Huynh-Feldt	4026.511	1.437	2802.305	3.109	.077

	Lower-bound	4026.511	1.000	4026.511	3.109	.096
HandgripDevice * Contraction * Sex	Sphericity Assumed	2439.571	3	813.190	1.884	.144
	Greenhouse-Geisser	2439.571	1.233	1978.946	1.884	.184
	Huynh-Feldt	2439.571	1.437	1697.853	1.884	.180
	Lower-bound	2439.571	1.000	2439.571	1.884	.188
HandgripDevice * Contraction * Order_Starting_Device	Sphericity Assumed	524.059	3	174.686	.405	.750
	Greenhouse-Geisser	524.059	1.233	425.109	.405	.574
	Huynh-Feldt	524.059	1.437	364.726	.405	.604
	Lower-bound	524.059	1.000	524.059	.405	.533
Error(HandgripDevice*Contraction)	Sphericity Assumed	22015.025	51	431.667		

	Greenhouse-Geisser	22015.025	20.957	1050.487		
	Huynh-Feldt	22015.025	24.427	901.274		
	Lower-bound	22015.025	17.000	1295.001		

7. FCU EMG (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
HandgripDevice	Sphericity Assumed	8681.593	1	8681.593	1.855	.191
	Greenhouse-Geisser	8681.593	1.000	8681.593	1.855	.191
	Huynh-Feldt	8681.593	1.000	8681.593	1.855	.191
	Lower-bound	8681.593	1.000	8681.593	1.855	.191
HandgripDevice * Sex	Sphericity Assumed	9890.471	1	9890.471	2.114	.164
	Greenhouse-Geisser	9890.471	1.000	9890.471	2.114	.164
	Huynh-Feldt	9890.471	1.000	9890.471	2.114	.164
	Lower-bound	9890.471	1.000	9890.471	2.114	.164

HandgripDevice * Order_Starting_Device	Sphericity Assumed	488.073	1	488.073	.104	.751
	Greenhouse-Geisser	488.073	1.000	488.073	.104	.751
	Huynh-Feldt	488.073	1.000	488.073	.104	.751
	Lower-bound	488.073	1.000	488.073	.104	.751
Error(HandgripDevice)	Sphericity Assumed	79546.582	17	4679.211		
	Greenhouse-Geisser	79546.582	17.000	4679.211		
	Huynh-Feldt	79546.582	17.000	4679.211		
	Lower-bound	79546.582	17.000	4679.211		
Contraction	Sphericity Assumed	658080.224	3	219360.075	1.318	.279
	Greenhouse-Geisser	658080.224	1.018	646511.620	1.318	.268
	Huynh-Feldt	658080.224	1.149	572915.244	1.318	.271
	Lower-bound	658080.224	1.000	658080.224	1.318	.267
Contraction * Sex	Sphericity Assumed	855364.706	3	285121.569	1.713	.176
	Greenhouse-Geisser	855364.706	1.018	840327.974	1.713	.208
	Huynh-Feldt	855364.706	1.149	744668.295	1.713	.208
	Lower-bound	855364.706	1.000	855364.706	1.713	.208

Contraction * Order_Starting_Device	Sphericity Assumed	51227.302	3	17075.767	.103	.958
	Greenhouse-Geisser	51227.302	1.018	50326.761	.103	.757
	Huynh-Feldt	51227.302	1.149	44597.758	.103	.787
	Lower-bound	51227.302	1.000	51227.302	.103	.753
Error(Contraction)	Sphericity Assumed	8490232.970	51	166475.156		
	Greenhouse-Geisser	8490232.970	17.304	490645.907		
	Huynh-Feldt	8490232.970	19.527	434792.680		
	Lower-bound	8490232.970	17.000	499425.469		
HandgripDevice * Contraction	Sphericity Assumed	47634.642	3	15878.214	.568	.639
	Greenhouse-Geisser	47634.642	1.056	45125.561	.568	.470
	Huynh-Feldt	47634.642	1.199	39739.636	.568	.490
	Lower-bound	47634.642	1.000	47634.642	.568	.461
HandgripDevice * Contraction * Sex	Sphericity Assumed	91203.158	3	30401.053	1.088	.363
	Greenhouse-Geisser	91203.158	1.056	86399.174	1.088	.315
	Huynh-Feldt	91203.158	1.199	76087.070	1.088	.323
	Lower-bound	91203.158	1.000	91203.158	1.088	.312
	Sphericity Assumed	7701.224	3	2567.075	.092	.964

HandgripDevice * Contraction * Order_Starting_Device	Greenhouse-Geisser	7701.224	1.056	7295.574	.092	.779
	Huynh-Feldt	7701.224	1.199	6424.817	.092	.810
	Lower-bound	7701.224	1.000	7701.224	.092	.766
Error(HandgripDevice*Contraction)	Sphericity Assumed	1425223.609	51	27945.561		
	Greenhouse-Geisser	1425223.609	17.945	79420.716		
	Huynh-Feldt	1425223.609	20.377	69941.521		
	Lower-bound	1425223.609	17.000	83836.683		

8. TB EMG (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
HandgripDevice	Sphericity Assumed	266.707	1	266.707	.771	.393
	Greenhouse-Geisser	266.707	1.000	266.707	.771	.393
	Huynh-Feldt	266.707	1.000	266.707	.771	.393
	Lower-bound	266.707	1.000	266.707	.771	.393
HandgripDevice * Sex	Sphericity Assumed	135.148	1	135.148	.391	.541
	Greenhouse-Geisser	135.148	1.000	135.148	.391	.541
	Huynh-Feldt	135.148	1.000	135.148	.391	.541

	Lower-bound	135.148	1.000	135.148	.391	.541
HandgripDevice * Order_Starting_Device	Sphericity Assumed	227.581	1	227.581	.658	.429
	Greenhouse-Geisser	227.581	1.000	227.581	.658	.429
	Huynh-Feldt	227.581	1.000	227.581	.658	.429
	Lower-bound	227.581	1.000	227.581	.658	.429
Error(HandgripDevice)	Sphericity Assumed	5531.950	16	345.747		
	Greenhouse-Geisser	5531.950	16.000	345.747		
	Huynh-Feldt	5531.950	16.000	345.747		
	Lower-bound	5531.950	16.000	345.747		
Contraction	Sphericity Assumed	6527.905	3	2175.968	.104	.958
	Greenhouse-Geisser	6527.905	1.501	4350.132	.104	.847
	Huynh-Feldt	6527.905	1.828	3570.127	.104	.886
	Lower-bound	6527.905	1.000	6527.905	.104	.752
Contraction * Sex	Sphericity Assumed	82251.959	3	27417.320	1.305	.284
	Greenhouse-Geisser	82251.959	1.501	54811.901	1.305	.282
	Huynh-Feldt	82251.959	1.828	44983.788	1.305	.284
	Lower-bound	82251.959	1.000	82251.959	1.305	.270

Contraction * Order_Starting_Device	Sphericity Assumed	27750.028	3	9250.009	.440	.725
	Greenhouse-Geisser	27750.028	1.501	18492.347	.440	.593
	Huynh-Feldt	27750.028	1.828	15176.555	.440	.630
	Lower-bound	27750.028	1.000	27750.028	.440	.516
Error(Contraction)	Sphericity Assumed	1008411.104	48	21008.565		
	Greenhouse-Geisser	1008411.104	24.010	41999.706		
	Huynh-Feldt	1008411.104	29.256	34468.899		
	Lower-bound	1008411.104	16.000	63025.694		
HandgripDevice * Contraction	Sphericity Assumed	648.250	3	216.083	.196	.899
	Greenhouse-Geisser	648.250	1.846	351.130	.196	.806
	Huynh-Feldt	648.250	2.337	277.386	.196	.854
	Lower-bound	648.250	1.000	648.250	.196	.664
HandgripDevice * Contraction * Sex	Sphericity Assumed	2219.143	3	739.714	.671	.574
	Greenhouse-Geisser	2219.143	1.846	1202.018	.671	.508
	Huynh-Feldt	2219.143	2.337	949.570	.671	.540
	Lower-bound	2219.143	1.000	2219.143	.671	.425
	Sphericity Assumed	3753.857	3	1251.286	1.134	.345

HandgripDevice * Contraction * Order_Starting_Device	Greenhouse-Geisser	3753.857	1.846	2033.309	1.134	.331
	Huynh-Feldt	3753.857	2.337	1606.273	1.134	.339
	Lower-bound	3753.857	1.000	3753.857	1.134	.303
Error(HandgripDevice*Contraction)	Sphericity Assumed	52947.537	48	1103.074		
	Greenhouse-Geisser	52947.537	29.539	1792.468		
	Huynh-Feldt	52947.537	37.392	1416.014		
	Lower-bound	52947.537	16.000	3309.221		

9. RPE (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Device	Sphericity Assumed	1.000	1	1.000	.332	.572
	Greenhouse-Geisser	1.000	1.000	1.000	.332	.572
	Huynh-Feldt	1.000	1.000	1.000	.332	.572
	Lower-bound	1.000	1.000	1.000	.332	.572
Device * SEX	Sphericity Assumed	1.365	1	1.365	.454	.510
	Greenhouse-Geisser	1.365	1.000	1.365	.454	.510
	Huynh-Feldt	1.365	1.000	1.365	.454	.510

	Lower-bound	1.365	1.000	1.365	.454	.510
Device * Device_Starting_Order	Sphericity Assumed	12.804	1	12.804	4.256	.055
	Greenhouse-Geisser	12.804	1.000	12.804	4.256	.055
	Huynh-Feldt	12.804	1.000	12.804	4.256	.055
	Lower-bound	12.804	1.000	12.804	4.256	.055
Error(Device)	Sphericity Assumed	51.148	17	3.009		
	Greenhouse-Geisser	51.148	17.000	3.009		
	Huynh-Feldt	51.148	17.000	3.009		
	Lower-bound	51.148	17.000	3.009		
Contraction	Sphericity Assumed	32.322	3	10.774	10.195	.000
	Greenhouse-Geisser	32.322	1.678	19.259	10.195	.001
	Huynh-Feldt	32.322	2.060	15.688	10.195	.000
	Lower-bound	32.322	1.000	32.322	10.195	.005
Contraction * SEX	Sphericity Assumed	16.947	3	5.649	5.346	.003
	Greenhouse-Geisser	16.947	1.678	10.098	5.346	.014
	Huynh-Feldt	16.947	2.060	8.226	5.346	.009
	Lower-bound	16.947	1.000	16.947	5.346	.034

Contraction * Device_Starting_Order	Sphericity Assumed	6.321	3	2.107	1.994	.127
	Greenhouse-Geisser	6.321	1.678	3.766	1.994	.160
	Huynh-Feldt	6.321	2.060	3.068	1.994	.150
	Lower-bound	6.321	1.000	6.321	1.994	.176
Error(Contraction)	Sphericity Assumed	53.894	51	1.057		
	Greenhouse-Geisser	53.894	28.531	1.889		
	Huynh-Feldt	53.894	35.024	1.539		
	Lower-bound	53.894	17.000	3.170		
Device * Contraction	Sphericity Assumed	1.377	3	.459	.500	.684
	Greenhouse-Geisser	1.377	1.753	.786	.500	.587
	Huynh-Feldt	1.377	2.168	.635	.500	.625
	Lower-bound	1.377	1.000	1.377	.500	.489
Device * Contraction * SEX	Sphericity Assumed	.915	3	.305	.332	.802
	Greenhouse-Geisser	.915	1.753	.522	.332	.692
	Huynh-Feldt	.915	2.168	.422	.332	.736
	Lower-bound	.915	1.000	.915	.332	.572
Device * Contraction * Device_Starting_Order	Sphericity Assumed	2.794	3	.931	1.015	.394

	Greenhouse-Geisser	2.794	1.753	1.594	1.015	.366
	Huynh-Feldt	2.794	2.168	1.289	1.015	.378
	Lower-bound	2.794	1.000	2.794	1.015	.328
Error(Device*Contraction)	Sphericity Assumed	46.800	51	.918		
	Greenhouse-Geisser	46.800	29.794	1.571		
	Huynh-Feldt	46.800	36.851	1.270		
	Lower-bound	46.800	17.000	2.753		

Sex* Contraction interaction

Tests of Between-Subjects Effects

Dependent Variable: RPE

Source	Type III Sum of Squares	df	Mean Square	F
Corrected Model	171.265[a]	7	24.466	3.749
Intercept	34953.340	1	34953.340	5355.699
Gender	52.740	1	52.740	8.081
Time	109.538	3	36.513	5.595
Gender * Time	3.763	3	1.254	.192
Error	992.010	152	6.526	
Total	36744.500	160		
Corrected Total	1163.275	159		

Tests of Between-Subjects Effects

Dependent Variable: RPE

Source	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[b]
Corrected Model	.001	.147	26.242	.975

Intercept	.000	.972	5355.699	1.000
Gender	.005	.050	8.081	.806
Time	.001	.099	16.784	.939
Gender * Time	.902	.004	.577	.085
Error				
Total				
Corrected Total				

a. R Squared = .147 (Adjusted R Squared = .108)

b. Computed using alpha = .05

Multiple Comparisons

Dependent Variable: RPE

Tukey HSD

(I) Time	(J) Time	Mean Difference (I-J)	Std. Error	Sig.
Contraction 1	Contraction 2	-.8875	.57124	.408
	Contraction 3	-1.6500*	.57124	.023
	Contraction 4	-2.2625*	.57124	.001
Contraction 2	Contraction 1	.8875	.57124	.408
	Contraction 3	-.7625	.57124	.542
	Contraction 4	-1.3750	.57124	.080
Contraction 3	Contraction 1	1.6500*	.57124	.023
	Contraction 2	.7625	.57124	.542
	Contraction 4	-.6125	.57124	.707
Contraction 4	Contraction 1	2.2625*	.57124	.001
	Contraction 2	1.3750	.57124	.080
	Contraction 3	.6125	.57124	.707

Multiple Comparisons

Dependent Variable: RPE

Tukey HSD

		95% Confidence Interval	
(I) Time	(J) Time	Lower Bound	Upper Bound
Contraction 1	Contraction 2	-2.3714	.5964
	Contraction 3	-3.1339	-.1661
	Contraction 4	-3.7464	-.7786
Contraction 2	Contraction 1	-.5964	2.3714
	Contraction 3	-2.2464	.7214
	Contraction 4	-2.8589	.1089
Contraction 3	Contraction 1	.1661	3.1339
	Contraction 2	-.7214	2.2464
	Contraction 4	-2.0964	.8714
Contraction 4	Contraction 1	.7786	3.7464
	Contraction 2	-.1089	2.8589
	Contraction 3	-.8714	2.0964

Based on observed means.

The error term is Mean Square(Error) = 6.526.

*. The mean difference is significant at the .05 level.

10. NRS-Pain (All Effects)

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Device	Sphericity Assumed	5.696	1	5.696	2.699	.119

	Greenhouse-Geisser	5.696	1.000	5.696	2.699	.119
	Huynh-Feldt	5.696	1.000	5.696	2.699	.119
	Lower-bound	5.696	1.000	5.696	2.699	.119
Device * SEX	Sphericity Assumed	.144	1	.144	.068	.797
	Greenhouse-Geisser	.144	1.000	.144	.068	.797
	Huynh-Feldt	.144	1.000	.144	.068	.797
	Lower-bound	.144	1.000	.144	.068	.797
Device * Device_Starting_Order	Sphericity Assumed	.681	1	.681	.323	.577
	Greenhouse-Geisser	.681	1.000	.681	.323	.577
	Huynh-Feldt	.681	1.000	.681	.323	.577
	Lower-bound	.681	1.000	.681	.323	.577
Error(Device)	Sphericity Assumed	35.870	17	2.110		
	Greenhouse-Geisser	35.870	17.000	2.110		
	Huynh-Feldt	35.870	17.000	2.110		
	Lower-bound	35.870	17.000	2.110		
Contraction	Sphericity Assumed	1.206	3	.402	1.019	.392
	Greenhouse-Geisser	1.206	1.998	.603	1.019	.372
	Huynh-Feldt	1.206	2.531	.476	1.019	.384
	Lower-bound	1.206	1.000	1.206	1.019	.327

Contraction * SEX	Sphericity Assumed	.285	3	.095	.241	.868
	Greenhouse-Geisser	.285	1.998	.143	.241	.787
	Huynh-Feldt	.285	2.531	.113	.241	.836
	Lower-bound	.285	1.000	.285	.241	.630
Contraction * Device_Starting_Order	Sphericity Assumed	2.243	3	.748	1.895	.142
	Greenhouse-Geisser	2.243	1.998	1.123	1.895	.166
	Huynh-Feldt	2.243	2.531	.886	1.895	.153
	Lower-bound	2.243	1.000	2.243	1.895	.186
Error(Contraction)	Sphericity Assumed	20.120	51	.395		
	Greenhouse-Geisser	20.120	33.969	.592		
	Huynh-Feldt	20.120	43.019	.468		
	Lower-bound	20.120	17.000	1.184		
Device * Contraction	Sphericity Assumed	.977	3	.326	1.212	.315
	Greenhouse-Geisser	.977	2.448	.399	1.212	.313
	Huynh-Feldt	.977	3.000	.326	1.212	.315
	Lower-bound	.977	1.000	.977	1.212	.286
Device * Contraction * SEX	Sphericity Assumed	1.380	3	.460	1.711	.176
	Greenhouse-Geisser	1.380	2.448	.564	1.711	.187
	Huynh-Feldt	1.380	3.000	.460	1.711	.176

	Lower-bound	1.380	1.000	1.380	1.711	.208
Device * Contraction * Device_Starting_Order	Sphericity Assumed	.707	3	.236	.877	.459
	Greenhouse-Geisser	.707	2.448	.289	.877	.443
	Huynh-Feldt	.707	3.000	.236	.877	.459
	Lower-bound	.707	1.000	.707	.877	.362
Error(Device*Contraction)	Sphericity Assumed	13.707	51	.269		
	Greenhouse-Geisser	13.707	41.615	.329		
	Huynh-Feldt	13.707	51.000	.269		
	Lower-bound	13.707	17.000	.806		

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